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ON THE ISOLATION FROM A CASE OF BRILL'S DISEASE OF A TYPHUS STRAIN RESEMBLING THE EUROPEAN TYPE*

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IN 1898, Dr. Nathan Brill[1] of New York reported on a disease which he observed in the wards of the Mount Sinai Hospital in New York and which, though having clinical resemblance to typhoid fever, never gave a Widal reaction. Seventeen cases of this condition occurred between 1896 and 1898, and a large number—something over 200—were studied up to December, 1909. Brill differentiated this fever, typhoid fever, and the other febrile conditions common to the region, and the disease was tentatively referred to as "Brill's disease". Later, it was found that the condition with which Brill was concerned was a mild endemic form of typhus fever. The clinical suspicion of this was subjected to experimental proof by Anderson and Goldberger in 1912[2]. They found that rhesus monkeys could be infected with blood from cases of Brill's disease, and that monkeys that had recovered from this infection were subsequently immune to Mexican typhus fever, and vice versa.

When the virus of Brill's disease was first studied, the differentiation of the Mexican type of typhus and the European variety had not been determined. This came later, with the observations of Neill[3], of Mooser[4] and of various students of the Rickettsia diseases, including ourselves[5]. In consequence of these recent studies, it has been further determined that the disease observed by Maxcy[6] in the South Eastern U. S. and studied by Dyer[7] and his associates and by ourselves is identical with the Mexican variety.

In another paper we have tabulated the well-marked differential characteristics which distinguish the European and the Mexican or "New World" types of typhus from each other[8]. We may briefly recapitulate these for present purposes, as follows:

Intraperitoneal injection of the Mexican virus into male guinea pigs is followed, in from four to seven days, according to the dose, by a sharp rise of temperature to from 104° to 106° F. At the same time, there is a marked scrotal swelling. This swelling, first noticed by Neill and first studied in detail by Mooser, is due to an inflammatory process of the tunica vaginalis, in the lining cells of which numerous Rickettsia can be found. Similar intraperitoneal injections of European virus give rise to scrotal swelling in exceptional instances only. The swelling is rarely marked, and Rickettsia are found in the cells in small numbers only. The temperature, with the European virus infections, does not rise until the eighth, ninth or tenth days. In the Mexican infection—possibly, as Mooser suggests, because of a preliminary immunization by the tunica process—the brain lesions are usually sparse and small; whereas, in the European cases, examined on the twelfth to the fourteenth days, they are profuse and well marked. These characteristics alone form entirely reliable distinguishing marks for the experienced student. In addition to these, however, there are further differences in the behaviour of the two viruses in rats, in the ease with which large amounts of Rickettsia can be obtained in prepared animals[9], and in the serologic and immunologic reactions of the respective causative agents[10]. These differences, which have been sufficiently described elsewhere, indicate the experimentally determinable existence of two closely related but none the less distinct forms of typhus virus. There are other types in addition to these, but for our present purposes we wish to deal only with the Mexican and the classical European forms.

The question arises whether the Brill's disease of the northern American cities, observed almost entirely among the crowded foreign-born populations (in a considerable percentage among the Hebrews coming here from South Eastern Europe, where typhus is endemic) represents the American or New World variety, or whether it is the European disease imported and kept alive among our city populations.

The question is of more than casual interest because of epidemiological considerations. From the studies of Dyer and his associates of the United States Public Health Service, as well as from those we carried out with the coöperation of Mooser in Mexico City[11], we have gained complete understanding of the cycle of transmission in the New World disease. We know that, in inter-epidemic periods, this virus survives in rats and is transmitted from rat to rat by the rat louse and the rat flea. From rats it can be passed to man by the rat flea, and epidemics occur when rat to man transmission has taken place in a community where general lousiness is associated with crowding and defective hygiene. That this form of typhus exists in rats in seaports along the Mediterranean basin was deter-

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mined by European investigators[12] following the observations made in this country and in Mexico. But so far as the classical continental European typhus is concerned, no intermediate animal host has so far been discovered. Rat studies made in Europe and in North Africa have been without result up to the present time* and we have, therefore, no precise information concerning the manner in which the European disease is kept alive in the often prolonged periods between epidemic outbreaks. The mild sporadic cases which occur during such periods are not sufficiently close together either in time or place to render likely a constant transmission chain from case to case. And the infected louse suffers as we do, and dies of its disease usually within two weeks. For want of a better explanation, therefore, it has been tentatively assumed that the European disease may be kept going either by the existence of human carriers or by the persistence of a constant trickle of mild and unrecognized cases. Neither of these views, however, is based on more than a desire to explain a situation for which no accurate experimental data are available.

It would be a definite step forward in the study of typhus epidemiology if we could determine with certainty whether the endemic disease of the north Atlantic coastal cities which was described by Brill conforms to the American or to the European type.

Opinions concerning the nature of the Brill virus have differed, but no definite proof one way or the other has been possible, because no virus obtained from cases of this kind has been studied since the time when differentiation of the types became possible. The suspicion that the Brill virus might be an imported European variety rested, therefore, largely upon its case distribution, and has been seriously questioned in recent years.

During the last three years, we have studied all the cases of suspected typhus fever which we have been able to observe in Boston. We have not seen many†, but there may, of course have been a considerably larger number of unrecognized ones—a possibility which is rendered likely by the usually mild nature of the disease, the frequent indistinctness of the rash, and the fact that the Weil-Felix reaction has not become a routine procedure in more than a few hospitals.

Until we studied the last case, of which we are about to give detailed data, we were unable to obtain a virus, largely because of the lateness in the disease at which we were able either to obtain blood or to put lice on the patient. In one case, however, blood taken on the twelfth day showed a strong Weil-Felix reaction and powerful agglutinations with our own Mexican Rickettsia suspensions and with the European louse vaccine of Weigl. The table recording these agglutinations gave us no differential data, but is interesting in showing that, in this endemic northern disease, Rickettsia of both types are agglutinated with a potency equal to that of the Proteus reactions.

TABLE I

	1/10	1/40	1/80	1/160	1/320	1/640	1/800
Proteus X 19	++++	++++	++++	++++	+++	±	
Mexican Rickettsia	++++	++++	++++	++++	++++	++++	+++
Weigl Vaccine	++++	++++	++++	++++	++++	++	

Agglutination of Proteus X 19, our own Mexican Rickettsia obtained from rats, and the European louse vaccine of Weigl in the serum of a Boston patient, Beth Israel Hospital, taken for us on the twelfth day of the disease by Dr. Rane.

In the following case, which forms the main subject of this paper, we were fortunate in obtaining a virus for study.

History of A. B. Typhus Case at the Beth Israel Hospital, Boston

(Service of Dr. Linenthal, who kindly placed this record at our disposal.)

The patient, a woman of 63, Jewish, was admitted to the Hospital on May 18, 1933, and died on May 24. The diagnosis was fever, cause unknown; and because of the deep coma in which the patient remained almost throughout the entire hospital course, suspicion of hemorrhage in the cerebrum was held. Physicians at the hospital suspected the possibility of typhus, which was not contradicted by the negative Weil-Felix reaction, since the disease had not lasted long enough to give rise to a Proteus X 19 agglutination.

History—Five days before admission, the patient began to complain of weakness with fever and headache, and showed a tendency to drowsiness, these symptoms continuing until admission. Previous history of patient had no bearing on present condition. On admission she was drowsy and semi-comatose.

Physical Examination—Systolic murmur; enlarged liver; blood pressure 140/70, and evidence of some peripheral sclerosis.

*Preliminary rat studies made by us in Boston during the winter of 1932 were entirely negative so far as a typhus virus was concerned, though they revealed the presence of rat-bite fever infections. These negative results, however, have no significance. In Mexico, while an epidemic was actually going on, 85 rats had to be examined before a virus was obtained. Without luck, it might require the examination of thousands of rats in places where the disease is as rare as it is in Boston.

†A clinical study by Drs. A. C. Ernstene and J. E. F. Riseman entitled "Endemic Typhus Fever in Boston. A Report of Ten Cases." New Eng. J. Med. 209:542 (Sept. 14) 1933, indicates the infrequency of the occurrence of typhus in Boston.

Laboratory Tests—Urine: trace of albumin; some white blood cells, few red blood cells.

Blood: moderate anemia; leucocytes on admission, 3500; later climbing to 6000 and 15,000, with normal differential.

Serology: agglutination for typhoid and paratyphoid, negative; Weil-Felix, negative.

Hospital Course—The temperature on admission was 103.5, and followed an irregular course, as given in the chart, until death; dropping occasionally to, or

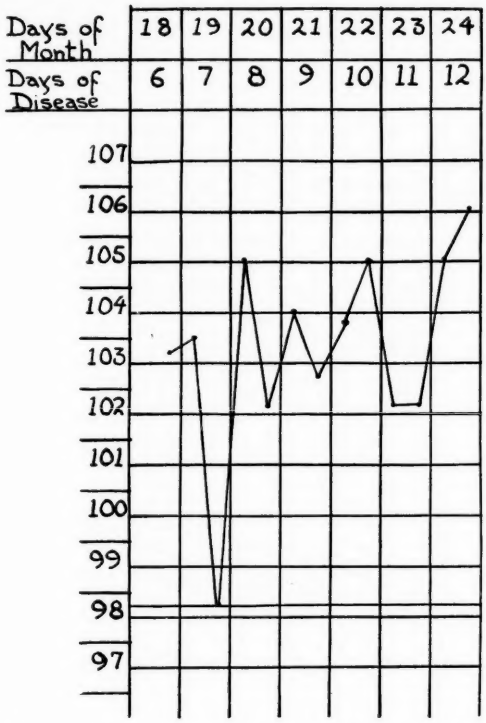

CHART I. Clinical chart of A. B., Beth Israel Hospital, Boston.

below 101°, but reaching 104° or over every day. The pulse was irregular, more or less following the fever.

Clinical notes are as follows:

May 18, 1933. Patient is drowsy, can be aroused only very little. Temperature 103.6, pulse 120, respiration 26. Skin hot and dry. No rose spots or petechiae. Outside of the systolic murmur and slightly enlarged liver, no definite signs of hemiplegia. No Babinski. No paralysis.

May 22, 1933. Moderate macular eruption of the skin noticed. Lumbar puncture gives no clues. The most prominent symptoms have been fever, coma and headache.

Since, among other things, the physicians of the Beth Israel Hospital considered the possibility of typhus of the Brill's disease type, the case was brought to the attention of the writers. Examination confirmed everything reported above, and it seemed that the possibility of severe typhus could not be excluded, although the rash was neither abundant nor entirely typical, and the Weil-Felix was negative. Blood was taken on May 22 and injected immediately into two guinea pigs.

EXPERIMENTAL DATA ON CASE A. B.

On May 22, at the bedside, 6 cc. of blood was injected into each of two guinea pigs. The history of guinea pig No. 1 is as follows: On the eighteenth day, the temperature rose above 104° F., and then fluctuated between 104.5° and 105° until the twenty-second day, when a brain inoculation was made from this animal to another guinea pig. The duplicate animal from the original blood injection was preserved for subsequent immunity test.

The guinea pig receiving brain material from animal No. 1 developed a typical temperature on the tenth day, rising on the thirteenth to 105.8°, when transmission was again made from this animal to three others, inoculations being carried out with spleen and blood and blood and brain, respectively. The chart of two of these animals, of the third and fourth passage generation respectively, is given in order to illustrate the temperature curve, which is typical of similar inoculations with European virus.

We refrain from burdening this paper with further charts, which all indicate the same thing. The virus is now in the twelfth animal passage and has caused invariably the same type of reaction in twenty-eight guinea pigs. Blood cultures have been taken in many of the animals and have been sterile except in a few instances, where organisms easily determinable as contaminations were found. Seven cultures taken from blood, spleen and brain of typically infected guinea pigs have been entirely free from bacteria. In one guinea pig only has there been any indication of scrotal swelling, and in this case—one of two animals injected in the fifth generation with brain—the swelling was slight and transient, a phenomenon which is observed with about the same frequency in typical European strains. Sections of the brain of guinea pig No. 8, taken on the fifteenth day, after seven days of temperature ranging between 104.2° and 105.4°, showed a large number of typical lesions of the type described by Otto and Dietrich[13] and subsequently studied by Wolbach and others[14]. These lesions, as has been stated, are much more profuse in the European variety than in the Mexican.

Cross immunity tests on convalescent guinea pigs have shown that the Boston virus protects against both the Mexican and European varieties of typhus infection, and that—vice versa—the European and Mexican convalescent animals are immune to the Boston infection.

SUMMARY AND CONCLUSIONS

To the student of typhus fever, the importance of our case A. B. lies in the fact that it demonstrates the existence in the larger cities of the northeastern seaboard of an endemic variety of typhus, the virus of which, in early animal pas-

sages, is indistinguishable from that of the classical continental European type. Proof of the fact that we are dealing with a typhus virus rests upon the nature of the infection produced in the guinea pigs injected with the patient's blood; on the subsequent characteristic temperature reactions produced in passage animals injected with brain, spleen and blood; on the development of the characteristic typhus brain lesions in such guinea pigs examined after the twelfth day; and upon the immunity of guinea pigs that have recovered from infection with the Boston strain to both the Mexican and the European types of infection, and vice versa.

We have used the expression "indistinguishable" in *early* animal passage from the European type, because many of the students of the various forms of typhus fever are at present considering the possibility that the two types of typhus fever with which we are here dealing are very closely related, and that the differences in the behaviour of the infectious agents obtained from them may be due to modification brought about by passage through different rodent hosts and insect vectors. This problem is one that is quite distinct from the purposes of the present report, and cannot be discussed in a brief communication with adequate accuracy. However, in regard to the first twelve guinea pig generations through which the Boston strain has been passed, it conforms entirely with the European variety and differs from the ordinary endemic strain isolated from fleas, rats or man in the United States and Mexico, the latter producing scrotal swelling, tunica Rickettsia and a more rapidly developing febrile reaction. It is true that occasional epidemic Mexican strains may fail to show scrotal swelling with the

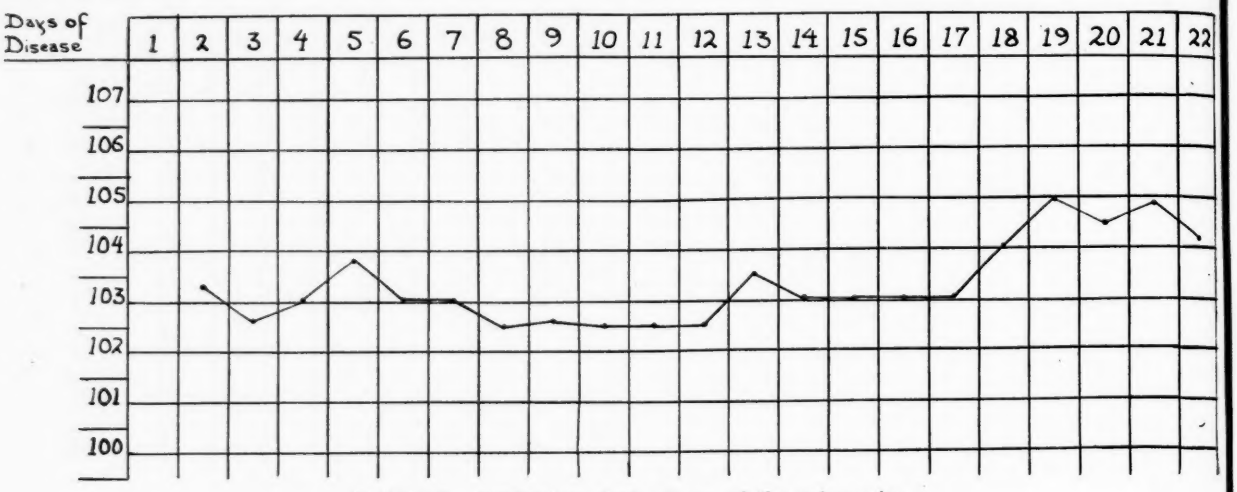

CHART II. Temperature chart of one of the guinea pigs injected directly with blood from the patient. Note the lateness of the characteristic rise of temperature.

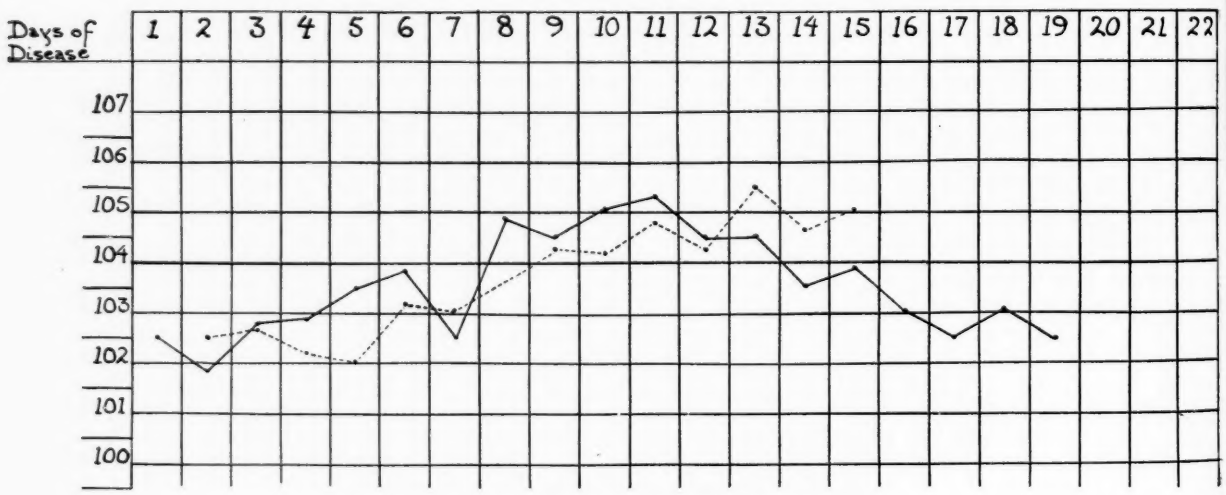

CHART III. Characteristic temperature reactions in two passage animals through which the strain from the Boston case was carried. The solid line is the record of Guinea Pig No. 5, one of the animals of the third passage generation. The broken line is that of Guinea Pig No. 8, an animal of the fourth generation.

regularity so characteristic of the New World virus, and the question of the possible existence of intermediate strains is now under active investigation in several places.

Of course this single case does not justify the assertion that Brill's disease is always caused by a virus of the European type, but it is of considerable importance to know that this type exists and appears to be permanently established in our larger cities. The fact that it almost invariably occurs among the foreign-born and especially among those coming originally from southeastern Europe renders reasonable the assumption that it is an imported European typhus.

The present study is of interest also in view of the opinion held by some observers that the so-called European type of infectious agent which causes a late temperature, profuse brain lesions and rare and negligible tunica lesions is associated with the epidemic form of the disease, while the type causing the rapid temperature, scrotal swelling, etc., is characteristically an endemic variety. This view, which we do not share, is to a considerable extent refuted by the behaviour of the strain described above. Of course, what this strain might do when passed through rats and fleas or grown in tissue cultures cannot be foretold; but it shares this uncertainty with the true European strain, which is being subjected to similar studies.

Of considerable importance for epidemiology and the problems of prevention is the fact that if in Brill's disease we are truly dealing with an imported form of European typhus, it may be possible by precise epidemiological studies of these cases to contribute important data and perhaps solve the hitherto uncertain problem of the manner in which the continental European virus survives during interepidemic periods.

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The Massachusetts Medical Society

PROCEEDINGS OF THE COUNCIL

Stated Meeting, October 4, 1933

A STATED MEETING of the Council was held in John Ware Hall, Boston Medical Library, Wednesday, October 4, 1933, at 12 o'clock noon. The President, William H. Robey, Suffolk, was in the chair and the following 156 Councilors present:

BARNSTABLE
W. D. Kinney

BRISTOL NORTH
W. H. Allen
A. R. Crandell

BRISTOL SOUTH
G. W. Blood
E. F. Cody
E. D. Gardner
W. A. Nield
I. N. Tilden
P. E. Truesdale

ESSEX NORTH
F. H. Coffin
E. S. Bagnall
R. V. Baketel
J. F. Burnham
H. F. Dearborn
A. P. George
T. R. Healy
E. P. Laskey
J. J. McArdle
F. W. Snow
L. T. Stokes
W. D. Walker

ESSEX SOUTH
R. E. Foss
F. W. Baldwin
N. P. Breed
J. F. Donaldson
W. T. Hopkins
J. F. Jordan
G. H. Kirkpatrick
A. E. Parkhurst
O. S. Pettingill
J. W. Trask

FRANKLIN
H. G. Stetson
A. H. Wright

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A. J. Douglas
G. D. Henderson
S. A. Mahoney
M. W. Pearson
G. L. Schadt

MIDDLESEX EAST
J. H. Blaisdell
Richard Dutton

MIDDLESEX NORTH
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G. A. Leahey
T. A. Stamas

MIDDLESEX SOUTH
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C. F. Atwood
H. T. Baldwin
E. W. Barron
C. F. K. Bean
E. H. Bigelow
G. F. H. Bowers
F. R. Clark
W. H. Crosby
A. C. Cummings
D. F. Cummings
A. W. Dudley
I. J. Fisher
N. M. Hunter
C. M. Hutchinson
A. M. Jackson
Josephine D. Kable
A. A. Levi
J. A. McLean
C. E. Mongan
F. L. Morse
Dwight O'Hara
W. D. Reid
M. J. Shaughnessey
C. H. Staples
H. P. Stevens
H. W. Thayer
W. S. Whittemore

NORFOLK
A. S. Begg
F. G. Balch
H. G. Batchelder
D. N. Blakely
W. L. Burrage
S. F. Curran
D. G. Eldridge
J. E. Fish
Alice M. Gray
J. B. Hall
F. E. Jones
G. W. Kaan
C. J. Kickham
M. J. Konikow
W. A. Lane
J. S. H. Leard
D. S. Luce
F. W. Marlow, Jr.
J. S. May
S. F. McKeen
Hyman Morrison
Benjamin Parvey
Victor Safford
D. D. Scannell
J. L. Sullivan
H. F. R. Watts

NORFOLK SOUTH
W. G. Curtis
F. L. Doucett
F. E. Jones
N. R. Pillsbury
C. A. Sullivan

PLYMOUTH
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T. H. McCarthy
J. J. McNamara
C. G. Miles
A. C. Smith

SUFFOLK
W. H. Robey
A. E. Austin
Gerald Blake
W. J. Brickley
C. S. Butler
David Cheever
F. J. Cotton
W. P. Cross
Lincoln Davis
R. L. DeNormandie
W. H. Ensworth
Reginald Fitz
Channing Frothingham
G. L. Gately
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W. R. Sisson
Louisa Paine Tingley
J. R. Torbert
H. P. Towle
F. A. Washburn
Conrad Wesselhoeft

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W. P. Bowers
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W. J. Delahanty
G. A. Dix
G. E. Emery
M. F. Fallon
J. J. Goodwin
David Harrower
E. L. Hunt
E. R. Leib
A. W. Marsh
J. W. O'Connor
E. H. Trowbridge
F. H. Washburn
S. B. Woodward

WORCESTER NORTH
T. R. Donovan
A. F. Lowell
H. R. Nye

On motion by S. B. Woodward, Worcester, duly seconded the reading in abstract of the record of the last meeting was dispensed with. The President thanked the Council for the honor conferred upon him by his election as president; he said he had visited several meetings of the District Medical Societies and planned to visit more soon; he thought that not enough was being done about the care of the indigent sick; that the program of the Society as to postgraduate medical instruction should be favored so far as possible; there were many important problems for consideration either by the Council or by its committees. He read the following obituary of a Councilor who had died since the last meeting:

Dr. John Hathaway Lindsey of Fall River died July 29, 1933. At the time of his death Dr. Lindsey was President of the Bristol South District Medical Society, and a Councilor of the State Society.

His premedical education was acquired in the public schools of his native city and in Brown University, from which he obtained his A.B. degree in 1892. He graduated in medicine from the University of Pennsylvania School of Medicine in 1899 and afterward studied in Berlin for two years.

He joined the Massachusetts Medical Society in 1904 and was also a Fellow of the American Medical Association. He was president of the Fall River Anti-Tuberculosis Association and was a member of the New England Roentgen Ray Society.

Dr. Lindsey is survived by his widow and four sons.

G. P. Reynolds, Suffolk, presented the report of the Committee of Arrangements for the Annual Meeting in Worcester next June. He and another member of his committee had visited Worcester and had consulted with the president and secretary of the Worcester District, and the officials of the Chamber of Commerce and it was the opinion that the new Auditorium was not available as a place of meeting because of its great size, lack of meeting places for the Sections and distance from the chief hotel, the Bancroft. Therefore the meeting would be in the Bancroft and in the adjacent Chamber of Commerce building. The large lobby of the hotel and the main dining room would be used for the commercial exhibits which are of interest to many of the Fellows and a source of payment of expenses of the meeting. There would be in addition scientific exhibits which would be well worth while. He moved that the days of the meeting be June 4, 5 and 6, 1934. The motion having been duly seconded, prevailed. The Secretary presented the report of the committee appointed in June to consider the petition of F. B. Worthington of Chatham to be restored to the privileges of fellowship, namely, that he be restored provided he pay within a month the dues for the current year. The report was accepted and its recommendations adopted by vote. A supplementary report of the committee to consider the petition of William Paris of Chelsea, recommending his restoration under the same conditions was adopted. Committee reports on the petitions of E. F. Prior of Cochituate, J. E. Dalton of Warren, M. G. Sullivan of Winchendon and J. J. McDevitt of Worcester, recommending restoration under the usual conditions were accepted. The report of the committee to consider the petition of I. F. Armstrong of Hudson who had been deprived of the privileges of fellowship by the Council, June 9, 1931 under the terms of Chapter I. Section 8, Clause (c) of the By-Laws, recommending that Armstrong be restored under the usual conditions, was debated and it was moved, seconded and voted to refer this report to the standing Committee on Ethics and Discipline, which had first recommended his deprivation, for a report to the Council. A new report of the committee to consider the petition for restoration of G. H. Schwartz of East Boston, recommending his restoration under the usual conditions was read by the Secretary. The chairman of the committee explained that they had given a hearing to the petitioner at which three physicians and a dentist appeared as witnesses together with the petitioner; that Schwartz had denied all charges against him and therefore the committee had voted to recommend his restoration. Voted to accept the report and adopt its recommendations. The Chair nominated and the Council appointed the following committees to consider the petitions for restoration of the following:

For O. A. Burton of Westminster, Vermont
Donald Gregg, H. W. Eliot, R. J. Carpenter.

For J. S. Walsh of Sunderland
Charles Moline, A. H. Wright, F. J. Barnard.

For F. E. Draper of Hyannis
G. H. Gray, C. E. Harris, J. I. B. Vail.

D. N. Blakely, Chairman, presented the following reports on Membership and on Finance, and they were accepted and their recommendations adopted by vote.

REPORT OF THE COMMITTEE ON MEMBERSHIP AND FINANCE, ON MEMBERSHIP

This Committee recommends:

1. That the following named six Fellows be allowed to retire under the provisions of Chapter I, Section 5, of the By-Laws:

1. Blake, John Bapst, Boston.
2. Butler, John Edward, Dorchester.
3. Churchill, Frank Spooner, Bass River, with remission of dues, 1932, 1933.
4. Jones, Elgin Wilbur, Lynn.
5. Patch, William Thurston, Newton Center.
6. Whitmarsh, Willard Francis, Bridgewater, with remission of dues, 1931, 1932, 1933.

2. That the following named three Fellows be allowed to resign under the provisions of Chapter I, Section 7, of the By-Laws:

1. Hemond, Fernard Joseph, West Warwick, R. I., with remission of dues, 1933.
2. Pierce, George Burgess, West Orange, N. J.
3. Plumb, Darley Garfield, Northampton, with remission of dues, 1931, 1932, 1933.

3. That the following named four Fellows be deprived of the privileges of Fellowship under the provisions of Chapter I, Section 8, Clauses (a) and (b), of the By-Laws:

1. Bill, José Penteado, Wayland.
2. Miranda, Carlos Alberto de la, Macedo de Cavaleiros, Portugal.
3. Pettengill, Warren Martin, Castle Point, N. Y.
4. Russell, Simon James, Springfield.

4. That the following named five Fellows be allowed to change their membership from one District Society to another without change of legal residence, under the provisions of Chapter III, Section 3, of the By-Laws:

One from Middlesex North to Suffolk

1. Drury, Dana Warren, Littleton.

Two from Middlesex South to Suffolk

1. Calicchio, David Julius, Allston.
2. Jackson, Henry, Chestnut Hill (Newton).

One from Suffolk to Middlesex East

1. *Daffinee, Ralph Weir, Melrose.

One from Suffolk to Norfolk

1. †Young, Edward Lorraine, Brookline.

DAVID N. BLAKELY, *Chairman.*

*Transferred to Suffolk, February 3, 1932.
†Transferred to Suffolk, June 9, 1914.

REPORT OF THE COMMITTEE ON MEMBERSHIP AND FINANCE, ON FINANCE

At the June meeting of the Council, the report of the Committee of the New England Medical Council contained a recommendation that the question

of an increased appropriation be referred to the Committee on Membership and Finance and it was so voted. The object of an increase would be to enable the Massachusetts Medical Society to assume a larger proportion than heretofore of the total expense of the New England Medical Council.

Our Committee has considered the information available and has also had the benefit of the individual opinions of the members of the Committee of the New England Medical Council and feels that it is inadvisable to ask the Council to make a special appropriation at this time or to offer any hope for an increased appropriation next year, therefore no action is recommended.

The Committee recommends that the assessment for 1934 shall be $10. for Resident Fellows and $6. for Non-Resident Fellows.

DAVID N. BLAKELY, *Chairman.*

The Chair nominated and the Council appointed the following Auditing Committee, to audit the Treasurer's accounts as of December 31, 1933: T. R. Goethals and J. W. Cummin.

E. F. Cody, Bristol South, ranking delegate from the Massachusetts Medical Society to the House of Delegates of the American Medical Association, took the floor to present a report of the six delegates to the meetings in Milwaukee, last June. He said that the full minutes of the House of Delegates had been published in the *Journal of the American Medical Association* in the issues of June 24 and July 1 and as 64 per cent of the members of the profession in Massachusetts subscribe for that journal it is presumed that Councilors are well informed on the topics considered by the House, therefore his report would be confined entirely to particular matters. (See Appendix.) The report was accepted by vote.

W. A. Lane, Norfolk, made the following motion and moved further that the matter be referred to the Committee on State and National Legislation. *Moved:* That the President in conjunction with the Committee on State and National Legislation be empowered at their discretion to appoint a legislative counsel who shall represent the Massachusetts Medical Society in such matters as may concern it before the General Court. The salary of such legislative counsel shall be approximately $600.00 per year or as may be determined by the Committee on State and National Legislation.

C. G. Miles, Plymouth, addressed the Council in seconding the motion. He was serving in the Massachusetts Senate as a representative of the medical profession; he knew the lack of influence of the profession at the State House; he thought a legislative counsel would help to improve present conditions, but the profession as a whole must assist in hearings on bills of medical purport. In the past very few physicians had appeared before the committees giving hearings; the legislative counsel must be a fighter in order to accomplish anything. Dr. Miles announced that he was going to do all he could to advance the interests of the profession on Beacon Hill but that he needed support.

Dr. Lane's motion was put, seconded and passed without dissent. F. J. Cotton, Suffolk, before introducing the following motion read a copy of his letter to the editor of the *New England Journal of Medicine,* issue of July 27, 1933, page 216, concerning a State Board to pass on all suits for alleged malpractice. He had talked the matter over with several prominent men and with the counsel of the Society and thought that the plan had met with approbation.

Moved, that the President appoint a committee of three to consider the advisability and possibility of using medical referees in cases of Malpractice suit, somewhat in the way that medical referees now pass on questions of sanity in criminal cases. The function of such experts would be not strictly judicial, but as advisors to the Court.

It would seem desirable to have all cases in which a Malpractice suit is brought, referred to such a board of experts, but it may, on consideration, seem more practicable to have this reference permissive, not obligatory. Such a board of Medical experts should, of course, be appointed by the Governor of the Commonwealth of Massachusetts, as is now done in the case of the experts who pass on sanity.

Further, be it moved, that the President of the Massachusetts Medical Society request the President of the Massachusetts Bar Association to appoint or designate a committee to confer with the committee above mentioned in the study of this problem and in formulation of any plan suggested for proposed legislation. The report of the joint committee to be submitted to the two societies for whatever joint action may seem best in the judgment of the Medical Society and the Bar Association.

The motion, having been duly seconded, prevailed.

The President appointed this committee:

F. J. Cotton
W. E. Ladd
L. E. Phaneuf.

E. F. Cody, Bristol South, *Moved* that the President appoint a committee of five to revise the By-Laws of the Society and to report at the next meeting of the Council. The motion was seconded and passed.

The President subsequently appointed this committee:

David Cheever
P. E. Truesdale
E. E. Tyzzer
A. S. Begg
W. L. Burrage.

E. S. Bagnall, Essex North, called attention to the fact that Alden B. George of Haverhill, admitted a Fellow last November, had failed to receive his diploma, sent him by first class mail. He moved that the Secretary be authorized to issue a duplicate certificate to Dr. George and that in future diplomas be sent to all new Fellows by registered mail. The motion was seconded by J. F. Burnham, Essex North, put to a vote and passed. M. J. Konikow, Norfolk, called attention to the fact that physicians have no special privileges as regards running their

automobiles in the streets of cities and towns and thought that they should be treated with right of way as are machines of the fire department. T. J. O'Brien, Suffolk, explained that this matter had been taken up with the authorities at the State House; that if the families of the physicians were to use their cars, in their absence, they would be subject to arrest; that there was no possibility of having any special privilege legislation passed. P. E. Truesdale, Bristol South, spoke of the importance of the work being done by the Committee on Postgraduate Medical Instruction; he thought it a missionary service that ought to raise the average medical attainments of the profession of the State and should have the hearty backing of the entire Society. The Chair said that he had visited several District Medical Societies and had found much interest in this postgraduate instruction; he would advance its interests whenever he could. Physicians having doubts about any of the courses should get into touch with Dr. Leroy E. Parkins, Secretary of the committee, although each District has its chairman. Information about the courses and the statistics of attendance will be published in the *Journal*.

Adjourned at 1.30 P.M.

WALTER L. BURRAGE, *Secretary*.

APPENDIX TO THE PROCEEDINGS OF THE COUNCIL

ABSTRACT OF REPORT READ OCTOBER 4, 1933

PROCEEDINGS, HOUSE OF DELEGATES AMERICAN MEDICAL ASSOCIATION, MILWAUKEE, JUNE 12-16, 1933

Your delegates attended every session of the House.

The Secretary of the Association reported the membership on April 1, 1933 as 97,111 names. During the year ended December 31, 1932 the names of 8,877 new members were enrolled, while 9,972 were removed from membership, of which 1,502 were the names of deceased members.

The number of names on the Fellowship roster on April 1, 1933 was 62,495. During the calendar year past 3,317 names were added while 5,259 were removed. The membership in Massachusetts was given as 4,814, the Fellows as 3,037.

The report of the Board of Trustees and of the Treasurer showed that the total income for the year was $1,634,848, less by $193,936 than in 1931. Total expenditures were $1,541,005, a reduction in expense of $58,125 as compared with 1931. At the end of the year the Association had invested in bonds the sum of $1,895,831. An appraisal of the investments on December 15, 1932 showed the depreciation had amounted to less than 6 per cent. The total reserve was $1,995,955. Net worth as at December 31, 1932, $3,400,706.

Of the publications of the Association, *The Journal* showed net earnings of $605,095; the health journal *Hygeia*, a margin above expense of production of $18,768.62. Of the eight special periodicals only two yielded any profit,—*The Archives of Otolaryngology*, and *The American Journal of Diseases of Children*. *The Quarterly Cumulative Index Medicus* decreased in circulation by 134. The loss in publication was $43,435.

The reports of the several Bureaus and Committees under the Board of Trustees showed constructive progress.

The Council on Medical Education and Hospitals in its report submitted the following resolution:

Resolved,

> That should the House of Delegates so order the Council is prepared to extend to other special fields of medicine the service which it has rendered in the fields of radiology and pathology to the end that members of the medical profession and others who may be concerned may be able to distinguish readily those who have received training in the various branches of medicine from those who are merely self-constituted "specialists".

The Council has taken this position for the following reasons:

There are so many self-appointed but unqualified specialists in the field that the public, the medical profession, the trustees of hospitals, and government officials are unable to determine who may be trusted to perform those services which require special skill.

Unless the American Medical Association shall undertake to make available lists of those who conform to uniform and approved standards in the various special fields of medicine, the multiplicity of other agencies attempting to perform this service will inevitably lead to great confusion on account of the diversity of methods employed. With state legislatures, state boards, state societies, and local and voluntary boards of every sort assuming to pass judgment on the qualifications of specialists, only chaos can ensue.

The Council already has experience in the preparation of lists of physicians specializing in pathology and radiology so that the extension of these activities to other branches of medicine is no new and untried venture, but merely the enlargement of an already well-established activity.

In the House Dr. Kopetzky of New York offered a resolution authorizing the Council on Medical Education and Hospitals to coöperate with the official examining boards and the Association of American Medical Colleges, the Federation of State Medical Boards, and the National Board of Medical Examiners through an advisory council hereafter to be set up by the Trustees and the Council. This and the foregoing resolution of the Council were reported out by the reference committee as follows:

Resolved,

> That the Council may contribute to these special boards by granting some form of recognition to those which now or hereafter maintain satisfactory standards of organization and procedure.

This resolution was adopted by the House.

Further the Council states there is reason to believe that at present not less than 2000 American medical students are enrolled in the medical faculties of Continental Europe. As foreigners these do not enjoy equal privileges with the native students, so their anticipated return to the United States after graduation constitutes a serious problem for our licensing boards and for the medical profession. On the initiative of the Council a number of informal conferences were held resulting in the adoption by the Federation of State Medical Boards of the United States of a resolution as follows:

> That no American student matriculating in a European medical school subsequent to the academic year 1932-33 will be admitted to any state medical licensing examination who does not before beginning such medical study secure from a state board of medical examiners, or other competent authority, a

certificate endorsed by the Association of American Medical Colleges or the Council on Medical Education and Hospitals of the American Medical Association, showing that he has met the premedical educational requirements prescribed by the aforementioned associations.

That no student, either American or European, matriculating in a European medical school subsequent to the academic year 1932-33 will be admitted to any state medical licensing examination who does not present satisfactory evidence of premedical education equivalent to the requirements of the Association of American Medical Colleges, and the Council on Medical Education and Hospitals of the American Medical Association, and graduation from a European medical school after a medical course of at least four academic years, and submit evidence of having passed satisfactorily the examination to obtain a license to practice medicine in the country in which the medical school from which he graduated is located.

It is expected that the enforcement of this regulation will exercise a wholesome restraint on the European migration of those less desirable students who fail to gain admission to the approved schools of the United States and Canada.

In view of the fact that the medical profession is already overcrowded with a surplus of 25,000, the Council deems it advisable that the Association enlist the coöperation of college and high school faculties in placing before their pupils such information as will enable them to select a career, and to invite support in bringing about a substantial reduction of enrollment of medical students.

During the year past, 688 hospitals and other institutions have been visited. From the register the names of 241 hospitals have been dropped and 190 added.

Conforming to instruction of the House of Delegates in 1930 the Council is conducting a study of the hospitalization of patients with nervous and mental disease. A partial report shows an exhaustive study of 631 institutions.

The House of Delegates adopted a resolution endorsing the Minority Report of the Committee on the Costs of Medical Care; a resolution requesting other medical organizations to declare publicly their opinions on general social, legislative, and economic relationships of medical practice only through approved channels of the American Medical Association; a resolution that the Trustees initiate and pursue activities and efforts to terminate misleading and misrepresenting radio broadcasting related to medicinal remedies; one providing for the Association to urge its constituent and component societies to devote at least one meeting annually to which the local bar associations may be invited for the joint discussion of psychiatric service in the administration of criminal justice and the treatment of the offender; another that the Association condemns the persecution of any individual on account of race or religion by any state or under any flag.

A resolution to create a committee for study of birth control was laid on the table.

The chairman of the Judicial Council presented a recommendation of the Council that the name of Dr. H. D. Pease be dropped from the roll of members of the Association because of unethical conduct. The recommendation was adopted.

Your delegates are happy to announce that the Committee on Awards in the Scientific Exhibit bestowed a gold medal on Dr. Elliott P. Joslin, and Herbert H. Marks for excellence of presentation of an exhibit illustrating the prevention of diabetes mellitus and certain of its complications.

EDMOND F. CODY, *Delegate.*

STUDIES ON TUMOR METASTASIS*

IV. Metastases of Cancer of the Stomach

BY SHIELDS WARREN, M.D.†

AMONG 1,059 autopsy cases of malignant disease selected from the material of the Huntington Memorial Hospital, the New England Deaconess Hospital, the Pondville State Hospital, and the House of the Good Samaritan 69 cases of malignant disease of the stomach were encountered. This frequency (6.7 per cent) is very low as compared with that found in larger series and is far below the incidence of cancer of the stomach and liver as given in the Massachusetts mortality statistics. With regard to the latter point, however, it must be remembered that a very considerable portion of the deaths ascribed to cancer of the stomach and liver are really due to malignancy in the liver, metastatic from other sites. A good deal of inflation of the incidence of gastric cancer is referable to this confusion. Probably the most important factor operating to give an abnormally low number of cancers of the stomach in my material is the fact that gastric cancer is rarely treated, as compared with other types of cancer, and is probably less frequently hospitalized. These cases are briefly summarized in Table I.

TABLE I

CASES OF CANCER OF THE STOMACH

	Total cases	Male	Female	Average age at death	Duration: Average before treatment	Duration: Average after treatment	Duration: Average for untreated	Duration: Total	Treatment: None or palliative	Treatment: Operative	Treatment: X-ray	Treatment: Combined	No. of recurrence	No. of metastases
Adenocarcinoma	40	20	20	58.3	9.8 mos.	4.3 mos.	1.4 yrs.	1.2 yrs.	36	4	—	—	1	7
Carcinoma simplex	19	7	12	60.3	1.5 yrs.	3.5 mos.	8.3 mos.	1.1 yrs.	16	3	—	—	—	6
Colloid carcinoma	7	5	2	58.3	1.1 yrs.	8.8 mos.	1.0 yrs.	1.3 yrs.	4	2	1	—	1	2
Malignant adenoma	1	1	—	55.0	—	—	6.0 mos.	6.0 mos.	1	—	—	—	—	1
Lymphosarcoma	1	1	—	75.0	3.0 yrs.	9.0 mos.	—	3.8 yrs.	—	—	—	1	1	—
Fibrosarcoma	1	1	—	35.0	—	—	—	—	—	—	1	—	—	—

Over half the cancers were classified as adenocarcinomas. The carcinoma simplex or undifferentiated carcinoma was the next most frequent type, with 19 cases. Colloid carcinoma was encountered seven times, and lymphosarcoma and fibrosarcoma once each. The criteria which we have used in subdividing the carcinomas on histologic grounds are relatively simple, and closely parallel those utilized in studying the malignancies of the large bowel[1]. There are many similarities in appearance and behavior among the cancers of the stomach and those of the large intestine.

Those tumors which showed well-formed alveoli in at least 25 per cent of the regions examined were classified as adenocarcinoma. This is a fairly inclusive group, ranging from well-differentiated neoplasms to somewhat anaplastic, though clearly alveolar tumors. The amount of stroma was not considered of sufficient importance to influence the classification. The other large group, the carcinoma simplex, or undifferentiated carcinoma, tends to be markedly anaplastic and less than 25 per cent of the tumor shows alveolar arrangement. These tumors again may be either medullary or scirrhous. The infiltrating cancer of the signet ring type, although considered by some as a subvariety of the colloid carcinoma, I believe to be more properly placed here.

The colloid or gelatinous carcinomas are those in which there is a considerable degree of secretory activity, in many places so marked as to show only mucinous secretion or small clusters of tumor cells, separated from their stroma, afloat in pools of mucus. In gross these tumors are bulky, infiltrating, and rather soft, with a peculiar watery translucence on section.

The malignant adenoma is similar to the form encountered in the large intestine. It is a polypoid tumor, infiltrating late, with anaplastic epithelium, usually only one cell thick, lining numerous irregular alveoli.

The sex distribution is remarkably even. A preponderance of cases in women would not have been surprising, as the autopsies from the House of the Good Samaritan were on female patients only, thus tending to weight the series. Of the 1,059 cancer cases, 60 per cent were in women.

*From the Laboratories of Pathology of the Harvard Cancer Commission and of the New England Deaconess Hospital.
†Warren — Pathologist, New England Deaconess Hospital. For record and address of author see "This Week's Issue," page 857.

The average age at death varies surprisingly little in the three major groups, only from 58.3 to 60.3 years. In these same groups the total duration of symptoms in the cases where definite information was available varies only 0.2 years, ranging from 1.1 years in the carcinoma simplex to 1.3 years in the colloid carcinomas. This short duration contrasts with an average duration of 3.1 years for cases of breast cancer[2].

Only nine cases of the entire group were operated upon. One case was treated by x-ray.

So far as these studies indicate, there is no correlation between histologic appearance of the primary tumor and the presence or absence of metastases or the extent of metastasis. Curiously enough, one-third of the more anaplastic tumors showed no metastases, as against one-sixth of the less anaplastic. Upon casual examination the size of the individual tumor cells and the relation of the malignant cells to their stroma appear to make very little difference in the behavior of the tumor. Detailed studies on this point are in progress.

The relatively restricted distribution of metastases is striking. This undoubtedly is in part due to the short duration of the disease and the location of the primary tumor being such as to cause death, rather than death being brought about by secondary changes in other organs.

Aside from the regional lymph nodes, most metastases lodge in the liver and on the peritoneal surfaces. After these the distant nodes, the pancreas, and the lungs are involved in order of frequency. Less than ten per cent of the cases metastasized to the lungs. Only nine instances of metastases above the diaphragm were detected. The details of the distribution of metastases are shown in Table II.

of metastasis to sites which were more or less widely scattered, in contrast to 61 to nearby structures. No true Krukenberg tumor was encountered, although there were three metastases to the ovary.

The relative restriction of metastases is of importance from a clinical standpoint. If metastases are not found in adjacent lymph nodes and in the liver at the time of operation, the chance of more distant involvement having already occurred is relatively slight. Owing to the short duration of these cases no satisfactory data on the distribution of metastases in relation to time have been obtained.

In contrast to the breast cancers, no definite evidence of invasion of blood vessels by tumor cells was found, although invasion of lymphatics was relatively frequent. Lymphatic channels carry the great bulk of metastases, although a number occur by direct transperitoneal implantation. Since none of the cases which had metastasized to bone showed pulmonary metastases, the tumor emboli, if blood-borne, must have passed through the lung capillaries without lodgment.

It may be that the figures of involvement of the pancreas are low, as in some instances it was impossible to tell whether the pancreatic involvement was due to direct extension or to metastasis. In such cases direct extension was considered to have been the mode of spread. It might not be amiss to distinguish between the peritoneal metastases and those to the intestine and in the gall bladder. If microscopic examination showed the serosa to be involved, metastasis was considered as peritoneal. If, however, the muscularis or the submucosa was the site of metastasis, it was considered as an

TABLE II

DISTRIBUTION OF METASTASES IN CARCINOMA OF THE STOMACH

	No. of cases	No metastases	Regional nodes	Distant nodes	Liver	Peritoneum	Pancreas	Lung	Intestine	Gall bladder	Bone	Pleura	Ovary	Diaphragm	Adrenal	Spleen	Vagina	Bladder	Uterus
Malignant adenoma	1	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
Adenocarcinoma	40	7	28	12	16	11	3	4	3	3	2	3	1	3	1	—	—	—	—
Carcinoma simplex	19	6	10	3	5	5	4	2	1	1	2	—	2	—	1	1	1	1	1
Colloid carcinoma	7	2	3	1	2	3	—	—	—	—	—	—	—	—	—	—	—	—	—
Total	67	16	41	16	23	19	7	6	4	4	4	3	3	3	2	1	1	1	1

There were only 18 sites of metastasis. There were four metastases to bone, two from adenocarcinomas and two from carcinoma simplex. Metastases to bone with cancer of the stomach are more frequent than is usually appreciated. These metastases were all of the osteoclastic type and probably were hematogenous. Omitting lymph nodes, there were only 22 instances

involvement of the organ itself rather than the peritoneum.

The one case of lymphosarcoma metastasized extraordinarily widely, involving the heart, kidney, and thyroid, among other unusual sites. The case of fibrosarcoma was disseminated to ten different sites, with heaviest involvement of the liver and lungs.

SUMMARY AND CONCLUSIONS

1. Based on a study of 69 cases, cancers of the stomach are relatively limited in the distribution of metastases.

2. Among the carcinomas, histologic groupings are of relatively little significance so far as the occurrence or distribution of metastases is concerned.

3. Involvement of the peritoneum is relatively more frequent in the colloid carcinomas than in the other types, although the figures are too few to be statistically significant.

4. If the regional lymph nodes, the peritoneum, and the liver are free from metastasis, the presence of metastasis elsewhere in the body is unlikely.

REFERENCES

1 Warren, S.: Studies on tumor metastasis. 3. The distribution of metastases in carcinoma of the large intestine. New Eng. J. Med. 209:167 (July 27) 1933.

2 Warren, S., and Witham, E. M.: Studies on tumor metastasis. II. The distribution of metastases in cancer of the breast. Surg. Gynec. Obst. 57:81 (July) 1933.

THE TRAINING OF INTERNS IN THE SOCIAL ASPECTS OF MEDICINE*

BY HARRY A. DEROW, M.D.,† AND ETHEL COHEN, M.S.†

THE training of interns, which provides for the development of proficiency in medicine and surgery, does not always give adequate consideration to the patient himself. The aim for a rapid turnover of patients in a teaching hospital may at times receive the disproportionate attention of the intern. In order to experience the greatest possible variety of medical or surgical cases in his limited period of ward service, his tendency is to discharge the patient promptly when the diagnosis is made and the treatment outlined. This tendency may serve ultimately to defeat the purpose of the intern, inasmuch as frequent readmissions of patients occur, when insufficient attention is given to the patient's background and ability to carry out recommendations for after-care.

A clinical picture of a patient was well described by the late Dr. Francis W. Peabody[1] as "not just a photograph of a man sick in bed; it is an impressionistic painting of the patient surrounded by his home, his work, his relations, his friends, his joys, sorrows, hopes and fears. . . . The difficulty is that in a hospital one gets into the habit of using the oil immersion lens instead of the low power, and focuses too intently on the center of the field."

An unmarried bookkeeper of thirty-four with congestive heart failure, who improves during a hospital stay may be discharged to his lodgings on the fourth floor of a tenement house with no facilities for ordinary home nursing care. What would he have gained from his hospitalization, if he were to return soon afterwards in a state of decompensation, because no attention had been paid to the environmental factors affecting his after-care?

A young woman of twenty-six, an office manager with severe psychoneurosis but without organic disease, works thirteen hours a day to carry out her employer's economy program. She is obliged to remain in this position as sole wage earner in a family of five. In addition to the usual household expenses, she carries the burden of mortgage and tax charges on an unmarketable house purchased by her three brothers as a family home before their marriage. Her life is a continuous round of work, inordinate responsibilities, anxiety over an elder mentally deranged sister, unrelieved by the leaven of friends, recreation or any of the emotional outlets of a normal young woman. What an economic waste her hospitalization would have been if only negative findings of extensive x-ray and laboratory studies had been considered! What benefit would the patient have derived from her hospital experience?

A mother, seriously ill on the ward, with pneumonia, aggravated by undernutrition and exposure in a damp, cold tenement, is obsessed with worry over her three small children. They are ill at home with influenza and have only the care of their father, an underemployed barber whose earnings of six dollars a week are terminated while he is at home. What are the chances for the patient's recovery, and what will be the probable consequences if she is discharged to an unheated house, with scant food and three small children needing care?

In order to give due consideration to these social elements in medicine, many hospitals have developed different types of organization in which, as a general rule, the intern plays only a minor rôle. The concept of training physicians in this aspect of medicine has been well recognized for many years[2]. The teaching of medical students along these lines has been carried on in a few teaching hospitals with small groups of men for some time and is gradually developing in more hospitals, but up to the present it has reached only a relatively small number of students. Few interns have had the advantage of this instruction during their student years. Inasmuch as the main purpose of the hospital training of the intern is the preparation for his later work in the practice of medicine or surgery, it is important at this stage

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†Derow—Assistant Physician, Beth Israel Hospital. Cohen—Director, Social Service Department, Beth Israel Hospital. For records and addresses of authors see "This Week's Issue," page 857.

to teach him adequately to consider the patient's present health problem in relation to his total life experience.

At the Beth Israel Hospital, Boston, opened as an acute general hospital in 1928, a plan was evolved which it was hoped would meet all the needs of the patient, as well as provide training for the intern in an appreciation of the social component in medicine. A description of this plan was published[3] after it had been in use for over two years. As this plan has been in successful operation for an additional year and a half, no longer regarded as an experiment but as an integral part of the intern's training, the publication of an account of the experience seems worth while at this time.

The internship lasts twenty and one-half months, and is divided into five periods of four months each, plus a two weeks' vacation. The interns are given increasing responsibilities under the close supervision and guidance of the resident physician and visiting staff, in each succeeding period of four months, as follows:

Period 1: He is engaged in all phases of laboratory work, the results of which he sees applied in the study of individual patients during the daily ward teaching visit. For the first time, he hears the visiting staff and senior intern discuss prognoses and after-care.

Period 2: As junior on the ward, he is responsible for histories and examinations of patients not assigned to fourth year medical students. He carries out special procedures in examination and treatment of ward patients. In taking histories, he secures social data, guided by an outline which is also used by the medical students and the senior intern. In the absence of the senior intern, he is in charge of all ward patients. He meets their relatives and may obtain information previously inaccessible to the senior. In the emergency ward, he is called upon to admit patients, many of whom are unsuitable for an acute general hospital. If the opinion of the junior intern as to the unsuitability of the patient for admission is confirmed by the resident physician, the junior intern secures the advice and assistance of a hospital social worker as to possible alternatives for the patient's disposition.

Period 3: The intern has charge of the medical private pavilion and pediatrics private and ward services. He takes histories and has to plan for the disposition of patients on the pediatrics ward service. This service is relatively small, but in effect is a miniature of the adult medical service. The experience derived from the medical and social study of these patients represents the intermediate step leading to his next duties, the more complete and intensive experience on the adult medical wards.

After the third period, the intern has a vacation of two weeks.

Period 4: As a senior intern, he is in charge of the male and female medical ward service, consisting of forty patients. He supervises and is responsible for the work of the junior and laboratory interns.

Period 5: The intern is occupied with outpatient work, consisting of alternate days in the medical and skin clinics. He also has the care of the small skin house service.

The gradual development of social insight on the part of the intern begins with his first period of training. The varied and closely supervised experiences of the succeeding periods constitute a preparation for the responsibility which the senior intern is given in the plan to be described.

As a result of the rotation of service, every four months, there is a change of senior interns receiving this training in the social aspects of medicine. Although there is this continuous change of interns, the resident physician remains a constant factor for two years. This is significant because of the part he plays in the teaching plan.

THE PLAN

The plan for this training is divided essentially into four parts:

1. The social history secured by the senior intern from the patient;
2. The diagnostic and prognostic data secured by the senior intern during the medical visit;
3. Presentation and discussion of all these factors during Medical Social Ward Rounds;
4. Subsequent discussion of selected problems by the intern and social worker with a member of the visiting staff on service.

1. *Social History*

In the course of the intern's study of each patient, certain basic social facts are elicited with the guidance of the following outline. The factors of personality, occupation and marital history are omitted from this outline as they are covered in other sections of the medical history.

Home Conditions

Dependent family or children without care
Emotional factors or conflicts
Type of home—neighborhood (describe)
  Tenement—number of rooms (adequate)
  What floor
  Suitability for convalescence

Economic Stability

Financial worries
Fear of losing job while ill

Factors for Consideration of After-Care

Ability to—
- Carry heavy housekeeping duties
- Understand and follow instructions
- Pay for special diet, if necessary
- Pay for appliance, if necessary

Need of—{Adjustment of occupation or school work
Convalescence outside home

Some of the information which cannot be secured from the patient during the first interview is obtained in subsequent contacts with him or his relatives.

2. *Diagnostic and Prognostic Data*

Factors in the Consideration of the Patient's Entire Problem:

"a) A statement of the specific problem to attain in each case.

b) Diagnosis and statement of the patient's physical and mental condition.

c) Probable duration of the patient's hospitalization.

d) Prognosis for immediate present and ultimate care.

e) Recommendations for after-care, e.g., convalescence, chronic hospital, home care, special diet, etc.

f) Statement of ability to resume activity or need for limitation: School, occupation, housekeeping."[3]

This last factor is discussed from the point of view of the patient's condition on his immediate discharge from the hospital, after recovery, or during the progression of the disease.

3. *Medical Social Ward Rounds*

The procedure is known as "Medical Social Ward Rounds" and occurs on the wards at a specified time each week. Those who participate in these Rounds are the resident physician, the senior intern and the social worker responsible for the service. On occasions, students of the School of Social Work, and fourth year medical students are also present.

The senior intern presents orally the factors listed above as they relate to the entire problem of each patient, taken up in consecutive order. As a result of the subsequent discussion between the senior intern, resident, and social worker, a variety of types of situations are encountered, as follows: Those which can be handled satisfactorily by patient or patient's relatives; those in which it has been possible to secure adequate medical and social data to state the problem and outline alternative plans of treatment; those which require further medical study and more consideration later on; those previously presented but now more completely worked up medically and socially; those with definite medical diagnoses but with no social complications apparent to the doctor. However, his statement of the social data may suggest to the social worker medical social problems requiring further study. Some of these patients who on previous Rounds presented no medical-social problems are now ready for discharge. However, when they are finally considered for discharge, the senior intern restates briefly the patient's medical condition and the social situation, and indicates the plan the patient, the patient's family or his friends have for his after-care. If, in the judgment of the social worker, the proposed plan seems unsuitable, the situation is discussed further for other disposition.

After all the patients have been presented in turn and discussed as outlined above, the senior intern, resident physician and social worker review all the cases which were considered to be in need of social study and treatment. Because of the limited number of social service personnel, it is not possible to handle all of the situations for which social service would seem desirable. It is, therefore, the responsibility of the participants in this plan to select those problems which present the greatest need for this service. During this process of evaluation and discrimination, the most valuable work has resulted to all the individuals involved.

In addition to the formal weekly Rounds the intern and the social worker have frequent informal conferences, because some of the information desired may become available immediately following one of the Rounds, and may contribute to the beginning of a plan for the patient, a change in a previous plan, or to the immediate discharge of the patient. In these same informal conferences, patients are referred who have been admitted to the hospital since the previous Rounds, and who may require immediate consideration. These conferences add to the flexibility of the relationship between intern and social worker and prevent delays in treatment which would result from complete reliance on the weekly Rounds.

The resident physician's contribution to this process of medical and social integration has been very important. He guides the intern in his general conduct of the Rounds, in the manner of presentation, and in the content of his material. When indicated, he contributes to the discussion from his wider medical and social knowledge. At times he modifies the senior intern's medical diagnosis and statement of prognosis. The experience of these Rounds impresses the resident with the importance of the patient as an individual so that he is enabled to teach social history taking to the fourth year medical students with the firm conviction that comes only from direct and personal participation.

4. *Relationship Between Visiting Staff and Medical Social Ward Rounds*

The medical diagnoses, prognoses and general questions of after-care are, of course, taken up by the visiting staff on the regular daily morning ward rounds with the interns in attendance. The data secured during these daily visits with

the staff physicians form the basis for the material which the senior intern presents at the Medical Social Ward Rounds. The visiting physicians are consulted by the senior and the social worker in special types of cases which comprise a relatively small proportion of the total number handled. They are classified briefly, as follows:

a) When the recommendation is made for a radical change in a patient's work or general way of life. For example, the head of a large family, owning a small bakery which he operates himself, has been advised to change his occupation.

b) When the medical social plan is not accepted by a patient seriously ill, and a safe compromise is needed.

c) When social plans involve large expenditures of money, either by patient with restricted income or by community, for chronic or convalescent care, or for change of climate.

d) When diagnosis is still in doubt, patient is ill, but does not require further acute hospital care, and home facilities seem inadequate.

e) On a small miscellaneous group of complex and delicate problems which require more mature judgment.

To those who have observed the plan in operation there have seemed to be many values derived from its use for the patient, the hospital, the medical social worker, and the doctor in training:

For the Patient:

1. Social study contributes to a better understanding of the patient.
2. Social adjustments are initiated promptly.
3. After-care is carefully planned in important cases, and social judgment given on all.

For the Hospital:

1. Period of hospitalization is generally shorter inasmuch as patients are referred early, the social plan is made promptly, and patients usually are discharged as soon as practicable.
2. Beds are available for a larger number of patients, as a result of the frequent turn-over."[3]
3. Less frequent readmissions of patients with exacerbations of chronic diseases.
4. Increased social awareness of entire ward staff of physicians and nurses.

For the Medical Social Worker:

1. She must be sufficiently articulate as to her function in the hospital and the underlying principles of social work to enable her to interpret them adequately to members of a profession trained to challenge ideas.
2. She must learn greater discrimination in the selection of problems, and to explain satisfactorily the reasons for her choice.
3. Through the orderly presentation by the doctor of medical facts and the discussions involved, the social worker has an invaluable opportunity to add to her medical knowledge.
4. The social worker's position as collaborator with the doctor becomes more clear through the demonstration of what she can contribute to the study and care of the patient.
5. The social worker's position in relation to the patient is strengthened in that he associates her as an integral part of his medical care.
6. From the point of view of the social service teaching, it enables her to demonstrate concretely in action to students from the School of Social Work the interrelation of disease and social maladjustment.

For the Doctor in Training:

1. A careful social history must be taken at the outset for presentation to his superior medical officer and a representative of another department. This experience requires him to be articulate about social factors and personality.
2. The methodical consideration of the social elements in medicine tends to develop an association of ideas in this direction.
3. He becomes familiar with various types of community resources for the patient's after-care, and learns how to evaluate them.
4. He is prepared to detect medical-social problems during his last period of training when in the Out-patient Department; to refer them to the social worker, and to avoid some unnecessary hospital admissions.
5. He is enabled to give more effective service later to patients in his private practice."[3]

The plan has provided for an orderly method of "refer" of patients to the social worker, and has served an additional and equally important purpose in the training of interns in

the social component of medicine. Twenty-four physicians and surgeons, nineteen interns and five residents, have already completed their hospital service with special emphasis on this phase of the care of the patient. All of the former interns, through their special interests, are distributed in a variety of clinical positions, where they may continue to care for patients along the lines of their hospital training. One of the former residents extends the influence of this training further, in his teaching of third year medical students and in the clinical contacts with physicians through his official hospital position.

Although from the day to day experience this plan has seemed satisfactory, a study is now in progress to test objectively the efficacy of recommendations as made by interns and residents on an individual case basis. All the problems referred on these Medical Social Ward Rounds during a specified period of time will be evaluated after a lapse of two or three years following the original recommendations.

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THE NEW HAMPSHIRE MEDICAL SOCIETY

ORTHOPEDIC TREATMENT OF INFANTILE PARALYSIS*

BY EZRA A. JONES, M.D.,† MANCHESTER

THE large majority of cases of Infantile Paralysis occur during epidemics, but considerable numbers are occurring outside of epidemics regardless of the time of year. This is a fact that we should keep in mind. At the clinics, patients are constantly coming in who show groups of paralyzed muscles which are unmistakably the results of infantile paralysis that have escaped the casual examination of the physician. Had there been an epidemic in the neighborhood, I feel sure that the physician and even the family would not have failed to recognize that the condition existed. These are the cases concerning which we should be constantly on guard, as valuable time is wasted before treatment is instituted.

Since the epidemic in New York during 1916, it has been pretty generally conceded that the so-called "orthopedic treatment" should be instituted at the start.

Convalescent serum in the preparalytic stage, and in the early paralytic stage, has been used with varying degrees of success, and Dr. Friborg has promised to take this up in his discussion. The most favorable reports by men who have been using serum, show a large percentage of residual paralysis. The fact that many paralytic cases go on to recovery with no treatment except rest in bed, makes it impossible to say just how valuable the serum treatment is. The fact that we are not able to tell which cases will recover and which will not, makes it necessary to follow out the treatment that we know has been successful. Orthopedic treatment should begin as soon as a diagnosis is made and should be continued until a definite prognosis can be decided upon.

As you know, this disease is due to an infection of the cells in the anterior columns of the cord, in the motor areas which preside over muscular activity. In the beginning these cells are in a hyperemic and toxic condition and they fail to respond to impulses sent from the brain. If these cells recover, muscular power is not lost, but if they die muscular paralysis ensues. It is fair to assume that the less irritation these nerve cells receive during this period of hyperemia the better are the chances of their recovery. That is why patients who have had immobilization of the affected parts at the onset of the disease show a larger percentage of recovery than those who are not held securely. I believe that any muscle pull or other motion of the affected parts sends an afferent impulse to an already damaged cell and keeps up an irritation which interferes with the chances of its recovery.

The length of time which is necessary to give these damaged nerve cells the best chance to recover, I think, is from six to eight weeks. Too long a period of immobilization is a waste of time and causes more muscular atrophy. While the muscles are kept at rest it is of the utmost importance that the affected members be placed in such a position that there will be no strain on them. A careful examination will determine the muscles or groups of muscles which are affected and we can tell exactly what the deformity will be. These parts should be placed in a position to prevent their being stretched either by gravity or by the opposing muscles.

The treatment can be divided into three stages.

1. Tender stage.
2. Convalescent stage.
3. Stage of residual paralysis and deformities.

The treatment should begin immediately after the diagnosis is made. Even in the first few

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days the weight of the bedclothes will stretch the anterior tendons, also placing of pillows under the knees will start flexion deformities in the knees and hips. Propping up of the patient in bed will stretch the back muscles and the patient has already started on to deformity. Many times there is acute muscle soreness and neuritis. Heat and perfect immobilization will usually relieve this. In some of the extreme cases, x-ray therapy has been used on the spine.

In the application of splints or whatever method of immobilization is used, a careful examination should be made to determine the muscles that have power and those that are paralyzed. This will be the guide as to the position the parts should be placed in. For instance, if the paralysis is in the upper extremities, and the shoulder girdle muscles are involved, the arm should be placed in abduction. This can be done either with a plaster spica, aeroplane, or wing splint. If the biceps is paralyzed, the arm should be placed in flexion; if both the biceps and triceps, a good position is about halfway between 90 and 180 degrees or 135 degrees. If the back muscles are paralyzed the patient should remain absolutely in the recumbent position and not be permitted to sit up. Probably the best means of immobilization of the spine is to use a plaster shell. This is moulded with the patient lying on his face with the spine hyperextended and should extend from just above the gluteal fold to the top of the shoulders. The shell should be padded with thick soft felt. The legs are best held by encasing them in plaster casts from the groin to the toes, care being taken that the foot is placed at right angles to the leg. If the paralysis is in the anterior group of muscles, the foot should be held in dorsiflexion; if the calf muscles are affected, the foot should be held in plantar flexion. These casts may be bivalved and, after the tenderness has disappeared, the upper half may be removed and light friction massage and heat can be applied to increase the circulation.

The second stage in this treatment is the convalescent stage. This begins after a period of from six to eight weeks, when all the tenderness has disappeared and some power has returned to the muscles. This is the time when muscle training and massage may be instituted. The patient should be encouraged to move the joint slightly, being careful not to fatigue the muscles. In some cases power will not have returned sufficiently so that any movement of the joint can be made voluntarily and in these the patient is helped to move it by an attendant. It is quite necessary that patients concentrate on this attempt to move the muscles.

Care should be taken not to remove the support too quickly, as weakened muscles and tendons stretch very easily and if the opposing muscles are very strong, they will stretch the weaker ones. By far the best method of restoring power to these weakened muscles is by exercise under water. This may be done in a bathtub or in a larger pool, if one is to be had. Patients who are unable to move a joint in the air can move it with ease when it is submerged in water. There is no doubt but that muscular activity is greater when the temperature is raised to slightly above the normal body temperature. Aside from the exercise patients are able to get in a warm bath, the ease with which they are able to move the weakened members is a source of great encouragement to them and helps to keep up their morale. When power has returned so that patients are able to get normal range of motion voluntarily, then it is safe to remove the splints.

At this time we come to the third stage. Now several months have elapsed and we are able to tell which muscles will probably not have return of power, and steps should be taken to prevent the occurrence of deformity by weight-bearing or antagonizing muscle pull. When the patient is able to get about, the weakened muscles should be protected by ambulatory splints. Into this class also fall the neglected cases where deformity has developed.

Deformity is caused, first, by the stretching of some group of muscles and contraction of the antagonizing group and by gravity and weight-bearing. Later the bony deformity occurs and must be taken into account. Braces are never expected to correct deformities. The function of the brace is simply to prevent deformity and before one is applied all parts must be placed in normal relations. After a period of from eighteen months to two years, the maximum of muscular power is usually obtained and now we may consider operative procedures. Of course this does not mean that all minor manipulations have to be postponed for two years, but it is probably better not to do any major corrections until about this time.

In general, there are three major types of procedure.

First, the purely manipulative type of operation in which with forcible manipulations contractures are corrected by the stretching of the soft tissues.

Secondly, open operations on the soft tissues. These consist of tenotomies, tendon lengthening, fasciotomy, capsulotomy and tendon transplanting.

Thirdly, operations to secure stability of parts which have become flail. These operations are to correct the deformity at the same time giving stability to the joints. These are cases of flail arms, feet in poor weight-bearing position and without sufficient muscle power to cause stability; and paralysis of the quadriceps muscles which necessitate the wearing of braces. In the latter cases, the flexors of the thighs, that is the posterior thigh muscles, had power enough so that it seemed advisable to transplant them

to take the place of the powerless quadriceps. Even a small amount of power in the quadriceps will greatly facilitate walking and obviate the necessity of wearing knee braces. If these muscles can be transplanted and power enough developed to fully extend the leg, the patient can walk with a great deal of security.

The history of orthopedic surgery has been a record of "repeated trial and error". For a time great possibilities were looked for in the field of muscle and tendon transplantation, but unfortunately the results over a period of years have been disappointing. However, the bone stabilization operation has been very satisfactory and we find that certain tendon transplants in selected cases are of great value. In connection with these bony operations, experienced judgment is necessary in the transposition of one muscle to do the work of another. This is especially true in the weight-bearing structures. In the arms and shoulders where tendons are transplanted, a weakened tendon may suffice, but in the legs and feet where there is constant strain for a long period of time many transplants have stretched and become of no value.

I will now show you three types of operative procedures which are quite simple and practical and the results of which are satisfactory.

Discussion

Dr. Carleton R. Metcalf, Concord: A few years ago, I was asked to go up to New London to see a boy who was suffering from infantile paralysis. He was in the paralytic stage. The boy had been working all summer as a caddy at Soo-Ni-Pee Park Lodge and there apparently had been no contact with infantile paralysis at all. I asked the doctor if he had had any other illnesses among children. He said, "Why, yes, a couple of weeks ago, there were seven or eight children at Soo-Ni-Pee Park Lodge with a stomach upset, and I gave them some castor oil and they all got well." I believe, gentlemen, that that doctor was confronted with a localized epidemic of infantile paralysis, and only one boy out of nine had developed paralysis. In other words, some people would tell you that practically everybody has infantile paralysis during childhood, and is, therefore, immune in adult life; that very few of these cases of poliomyelitis develop actual paralysis.

And so it is fair, in summer when the disease is prevalent, if you find a child who has a stomach upset which is very often the initial symptom, to bear in mind the possibility of a case of infantile paralysis. The clinical symptoms of nervousness and irritability should make you suspicious, and if you have the inclination to do a lumbar puncture so that you can pick up the case in its pre-paralytic stage, you should do it. It is probably true that, in New Hampshire, cases of this disease are almost never diagnosed in the pre-paralytic stage.

So far as the initial treatment goes, the important thing is rest and the avoidance of meddlesome therapeutics. Don't try electricity. Don't try counterirritation. Don't let the family argue you into having an osteopath or chiropractor. What the child needs is absolute rest, the avoidance of undesirable therapeutics and fixation of the affected joints in order to avoid deformity and muscle stretching and foot-drop, adduction of the shoulder, scoliosis, and other conditions. And remember, too, that in many of these cases, the muscles of the back and of the abdomen are paralyzed. Put the abdomen or the back, as the case may be, at rest. Later on, of course, we get the muscle training that Dr. Jones spoke to you about. It is amazing how much recovery a child may have simply by rest and muscle training, and how often a child who has had initially complete paralysis will come back and develop a useful extremity.

So far as the convalescent serum goes, I think most of us feel that it probably isn't much good.

Dr. Carl R. Friborg, Manchester:

The Value of Convalescent Serum in the Treatment of Poliomyelitis

Believing that members of this society would be interested in the value of convalescent serum in the treatment of poliomyelitis, I have reviewed considerable, available literature on this subject. I also had an opportunity to discuss this subject with Dr. Aycock of the Harvard Infantile Paralysis Commission, Dr. Philip Wilson and others who have studied and made an estimate of the value of this form of treatment.

In passing I might just mention that two other methods of treatment are the following:

1. The use of an antistreptococcus serum.
2. Because of the belief that the injury to nerve cells is secondary to edema and pressure on the spinal cord, the use of lumbar puncture and drainage is indicated.

The use of convalescent serum dates back to 1915 and was first employed by Netter in France. He administered the serum to patients who were paralyzed at the time of treatment. As paralysis is due to complete or partial nerve cell destruction, the treatment should be given during the pre-paralytic stage, which is usually during the first three days of the illness.

Briefly let me state some of the factors which make an estimate of the value of serum difficult to determine.

1. Whether or not a correct diagnosis was made.
2. The difference in therapeutic strength of serum collected from different donors.
3. Our inability to foretell those cases which will show paralysis and those which will not.

DOSAGE USED

Ordinarily the amount of serum used in each case is between 60 and 100 cc., 20 to 40 cc. intraspinously and the remainder intravenously.

RESULTS

During the summer of 1931 two extensive outbreaks of poliomyelitis occurred at about the same time; one in Brooklyn, New York, and the other in Hartford, Connecticut. Through the coöperation of two large hospitals, one in each city, the Harvard Infantile Commission was able to make an intensive study of the results obtained by the use of serum. Eighty-two cases of poliomyelitis were studied in the pre-paralytic stage, half of which received human convalescent serum by the intravenous and intraspinous routes.

Briefly the procedure was as follows: On admission a careful history was taken. If the duration of the illness was less than three days, the patient was accepted for study. A careful physical examination was made including the testing of all the muscle groups to rule out any detectable paralysis. Each case had a diagnostic lumbar puncture made. The dosage of serum given each case was 60 cc. intravenously and 20 cc. intraspinously followed in eight to twelve hours by 20 cc. additional intraspinously.

A second muscle examination was made 24 to 48 hours after admission and again before discharge, in most cases two to three weeks later.

Of the eighty-two cases studied forty-two or fifty-one per cent developed some paralysis divided about equally between the two cities. There were two deaths from respiratory paralysis. Both of these were in the treated groups, one in Brooklyn and one in Hartford. In the remaining cases the average paralysis for the treated groups was 12.1 per cent and for the untreated group 5.9.

Analyzed separately, the average paralysis per case treated in the Brooklyn group was 1.4 and in the untreated case 8.1.

In the Hartford group, however, the average paralysis in the treated cases was 20.4 whereas the untreated cases showed an average paralysis of 4.3. This difference between the two series is due to the inclusion of a relatively small number of severely paralyzed cases in the Hartford group.

Thus, in Brooklyn, when the treated cases are compared with the parallel untreated cases, the weight of evidence is with the treated group, whereas in Hartford, the results are reversed.

By these two controlled tests it is impossible to definitely say that convalescent serum is effective. However, on the other hand, after studying the Brooklyn figures, it is difficult to say that serum is of no value. Therefore, in my opinion, the use of convalescent serum at the present time in the treatment of poliomyelitis is of uncertain value.

DIAGNOSIS AND TREATMENT OF MULTIPLE SCLEROSIS*

BY PAUL DE NICOLA, M.D.,† NASHUA

MULTIPLE sclerosis is a degenerative disease of the nervous system, first described by Cruveilhier in 1835 and later more thoroughly classified by Valentine in 1856, but the famous triad of signs, namely, nystagmus, scanning speech, and intention tremor, were more accurately established in 1862 and 1863 by Charcot and Bouchard. These three signs have become pathognomonic of the disease in its classical form. In the less typical cases with which we come in daily contact, however, one or more of these may be absent. Then, there are the atypical cases that give us the most trouble in diagnosis.

Geographically, the disease has no preference for any particular locality, although it is said that it is more prevalent in Europe, but, according to Taylor, the reason for this is that is better diagnosed in Europe than it is in this country. Males seem to have a predisposition for the disease according to Stevens, and in my practice 85.7 per cent of the cases have been in males, thus justifying the opinion of many that it is more prevalent in the male. The ages vary from 10 to 25, and 15 to 41 years of age. (Stevens and Spear respectively.) But it is generally accepted that practically no age is exempt, although it is essentially a disease of young adult life.

The pathology of the disease is essentially a degeneration of the myelin sheath with more or less preservation of the axis-cylinder. The lesions are well disseminated in the cord, more particularly in the white matter. This condition seems analogous to a condition existing, although more rarely, in the brain, called encephalitis disseminata. A description of this is found in the *Journal of Neurology and Psychiatry*, November, 1932.

Much has been written of late years about the cause of multiple sclerosis. Infections, trauma, heredity, predisposition, endotoxins, exotoxins, and chemicals, such as lead, arsenic, and many other factors have been considered as causative agents of the disease. A great deal has also been said about a special spirochete isolated by

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†De Nicola—For record and address, see "This Week's Issue," page 857.

Purves-Stewart, and Kathleen Chevassut. (Taylor.) But this has been awaiting more definite confirmation. In a letter from Berlin in the *Journal of the American Medical Association* of March 4, 1933 (page 675), the question of infection was discussed from the standpoint of spinal fluid changes, the paroxysms of the disease, and the fact that a spirochete was isolated in ten cases out of forty-eight. Also, in a case in which the patient died in a second acute attack of the disease, there were found foci of degeneration in which spirochetes were seen within or on the edges thereof, that is the demyelinated areas.

Professor Sachs in this same letter gives us the result of a complement fixation test in multiple sclerosis with multiple sclerotic patients and controls which tend to give evidence of antibodies formed in this disease; but the findings are not constant and therefore not conclusive. Perhaps the stages of the disease must be taken into consideration as there may be antibodies formed during the exacerbations of the disease and none during the remissions. However, according to the percentages given, a characteristic reaction of the serums of the multiple sclerotic patients seems evident and the presence of specific substances of this disease in the serums of brains of patients is assumed.

Heredity, no doubt, plays a rôle in this disease as in other degenerative diseases of the nervous system. Curtius of the University of Heidelberg published statistics, reprinted in the *Journal of the American Medical Association,* May 6, 1933 (page 1471), that show there is a neuropathic tendency in the families of multiple sclerotic patients. Neuropathic disorders in the family and relatives of patients with multiple sclerosis, according to this investigator, are found two to three times as often as in the normal population in a given locality. These neuropathic disorders were distributed practically over the entire field of mental and nervous disorders recognizable. I have observed in all my cases practically the same neuropathic tendencies as Curtius found, and I think more and more that multiple sclerosis is not of an inflammatory origin from infections, but probably from hereditary tendencies toward nerve tissue deterioration.

In two of my cases in which the disease seems so far arrested after a period of four and five years respectively, abscessed teeth had been found and removed, but how much of a rôle they played in this disease, is not known.

DIAGNOSIS

There is no disease in existence but what has its atypical forms, and multiple sclerosis is no exception. Therefore, we must consider both the classical and non-classical ones. In all typical cases the famous triad of Charcot is present; namely, intention tremor, scanning speech, and nystagmus. One of the earliest symptoms of the disease is a fleeting ocular palsy accompanied by transient weakness in a limb or both limbs and some vesical disturbance. These may take place long before the definite final symptoms of the disease come on. The patient will first complain of weakness of a limb or of both lower extremities, with difficulty in walking, and may complain of dizziness. He may get a little emotional. It is at this stage of the disease that the erroneous diagnosis of hysteria is often made. Pain is not a feature of this disease at this stage or any stage. In about 40 per cent of my cases, the symptoms began in one lower extremity. Then, it is likely that these early symptoms will disappear and the patient thinks himself well or nearly well, only to have a sudden relapse and the symptoms come back with renewed vigor and become accentuated. The gait becomes spastic and ataxic with weakness, especially in the affected limb; and in walking this leg is dragged. The ataxia involves the upper extremities with signs of incoördination. The deep reflexes become exaggerated, and there may be a clonus and a Babinski. There is a disturbance of sensation in the lower extremities, and possibly in the upper, and about the trunk.

On account of the pyramidal tract involvement, the superficial reflexes become first decreased and then completely abolished. This seems to be typical of most cases. In fact, all of my cases showed absence of the superficial reflexes. The tremor is very characteristic, and in some cases becomes violent, but of course, only when the muscles are brought into play. Sometimes there is a marked tremor of the head and also marked speech disturbance when the vocal muscles, or those of the head are used. While the body is at rest or during sleep, tremor is entirely absent. During the focusing of the patient's eyes on an object, there is oscillation which apparently has the same source as other tremors of the body. The speech is so interrupted that words come out in staccato form and are pronounced in syllables. About 70 per cent of my cases showed speech disturbance, and speech disturbance seems to be more common in men than in women in my cases. Speech is sometimes explosive.

In the same fashion, writing is interfered with by the tremor, and a characteristic writing, where the contour of each letter is broken up, is formed, resulting in a sort of hen-scratch writing.

A certain amount of mental deterioration keeps pace with the disease, and, after a period of years, I find it present in about 70 per cent of the cases. Bladder disturbance was

present in 29 per cent of my cases, affecting men and women alike, and seemed to appear early. Stevens says it is present in four-fifths of all cases. Hippus is present in many cases. It is said that hippus is more present in multiple sclerosis than in any other disease. (*Journal of American Medical Association,* Feb. 25, 1933 [page 625]).

Intention tremor, although not present in all cases, is a very important symptom. A veteran whom I examined, and who was completely disabled, showed the tremor coming on as early as a month after what I supposed was the beginning of the disease. In August, 1919, during one of the last battles of the World War, this veteran was seized with an hysterical attack in which he screamed and could not control himself. This was followed immediately by a severe headache with nausea and vomiting. It only lasted a short time and he then continued to do his work until he was discharged the following November. His records showed, however, that in September, which was a month after he was seized with the above symptoms, he signed a paper for government insurance and later in November signed his discharge. Again, the next year he signed other papers, and the signatures, beginning from the one in September, bore an increasing demonstration of the writing of multiple sclerosis. His signatures until 1925 kept getting worse, until his writing was illegible.

The examination of the spinal fluid is also of help in diagnosis. It shows an increase in the lymphocytes; protein content is 0.04 per cent or less, globin is in excess, and gold soluble curve is of a paretic type, with a negative Wassermann. (S. A. K. Nelson.) The gold soluble reaction is said to be demonstrated more frequently in acute relapses and in acute retrobulbar neuritis. It is said to be negative during the remissions of the disease. (*Journal of Neurology and Psychiatry.* London, 1933.)

From hysteria we can differentiate this disease by the fact that nystagmus, tremor, sphincter disturbance, and optic atrophy are not present in hysteria.

The above symptoms of multiple sclerosis are absent in lateral sclerosis, and there are no remissions in the latter.

In locomotor ataxia, the positive Wassermann, the stamping gait, pupillary disturbances, loss of the deep reflexes, and a tendency to progress without remissions, differentiate it and multiple sclerosis.

In the case of tumors, there is persistent choked disk, increased intracranial pressure and a gradual tendency of the disease to progress without remissions. The tremendous headaches and projectile vomiting also differentiate this condition and multiple sclerosis.

TREATMENT

Owing to the uncertainty of the cause of this disease, no specific treatment has as yet been found. All treatment is symptomatic. As a specific treatment for the supporters of the theory of a spirochete and for treatment on general principles, arsphenamins have been used. Some report improvement, and some do not. Early, there seems to be a slight improvement, as demonstrated in about 30 per cent of my cases, but I have always felt that it was during the remission of the disease, as later the symptoms became aggravated and the end result showed no definite improvement. Arsphenamins were given in courses of eight intravenous injections and these were alternated with mercurial injections. Strychnin pushed to its physiological limits is used very extensively, and in my practice has been as satisfactory as any. Potassium iodide in mammoth doses also seemed to be of some benefit, especially in one case where the disease had been in progress for two years. It has been apparently arrested for the last four years and the patient has been attending his occupation daily. Malarial vaccines have been reported, but with no result. Typhoid vaccines have been used quite extensively, and also by the writer in two cases, with no result. Forced drainage of the spinal fluid has been found of value in the relief of such symptoms as amblyopia, Babinski and clonus due to reduction of intracranial pressure and the lowering of the osmotic tension of the blood. (Retan, George M.: Some clinical applications of forced drainage to various infections in inflammatory conditions in the central nervous system. Society Transactions of the New York Neurological Society, October 4, 1932. *Arch. Neurol. & Psychiat.* 29:404 [Feb.] 1933.) I have tried this on two or three cases, but the reaction was so great that severe headaches, nausea, vomiting, loss of weight, and even symptoms of collapse followed, that I immediately discontinued it. Specific protein therapy has also been used with poor result. Liver therapy has been used with no result. Sodium amytal, hyoscin hydro-bromide, veronal, and luminal are of value in cases with tremor. Hydrotherapy and massage of the affected limbs with heat, offer some relief from sensory disturbances. And finally, good nutrition and elimination are imperative. Removal of questionable foci of infection, such as teeth, tonsils, etc., is always advisable.

I wish to express my thanks to Dr. J. J. Thomas of Boston for his coöperation in supplying me with material for two cases that we both had seen at different times.

DISCUSSION

DR. DEERING G. SMITH, Nashua: We have listened to a comprehensive paper on an interesting disease which is often not diagnosed un-

til it reaches the advanced stage. The symptoms of this disease depend upon the location of the lesions, and these may be anywhere in the cerebrum or spinal cord. Transient numbness, weakness, paralysis, diplopia, or dimness of vision, occurring suddenly in a young adult should always make one suspicious of multiple sclerosis. The absence of abdominal reflexes is of great importance in helping to diagnose it. A man whom I saw late in the disease thought at first that the weakness of his legs and uncertain gait were due to an operation for appendicitis which preceded the onset of his difficulty in walking. A young lady believed that her dimness of vision, which came on suddenly while in college, was due to excessive studying. This patient had several long remissions, one of them lasting about twenty years. She taught school for thirty-five years, her only difficulty being a weakness of one leg and an uncertain gait. These long remissions are fairly common, and for that reason every effort should be made to encourage the patient and to keep him at his usual work, even though the final outcome is grave.

Dr. Charles H. Dolloff, Concord: Dr. deNicola and Dr. Smith have covered this disease and its various characteristics very well. It hardly seems as though I could bring any particular contribution to the discussion.

Even though we receive only few cases of multiple sclerosis from time to time at our hospital, I am interested in this disease. The number of these cases is small, perhaps two or three a year. When they do come to us, they are apt to be rather advanced cases.

One of the interesting phases of this disease, so far as the psychiatrist is concerned, is the possibility of its being mistaken in its earlier stages for hysteria.

I am particularly interested in the war case reported by Dr. deNicola, in which the patient seemed to develop multiple sclerosis as a result of fright. Some of our cases have the condition following trauma, but we are inclined to think that this is an attempt on the part of the patient to explain the cause rather than that it is actually due to trauma.

I do not find that there is any treatment yet which offers any real hope for this disease. It is an interesting disease because the pathology is so definite, the etiology so obscure, the symptoms so vague, and treatment so hopeless.

I believe the latest treatment is the use of quinine in the very early stages, and this has offered some hope, but thus far, neurologists have not supported this to a great extent.

The draining of the spinal fluid I understand is not offering much help. I was much interested in what Dr. deNicola said about the spinal pressure. I have understood that these cases do not show increased spinal fluid pressure, and that that was one of the diagnostic differentials of multiple sclerosis and luetic conditions.

Dr. Simon Stone, Concord: I was also interested in Dr. deNicola's remarks about the spinal fluid findings in multiple sclerosis. He mentioned the use of continuous drainage of the spinal canal as a means of relieving the increased intracranial pressure. As a matter of fact, the spinal fluid pressure in multiple sclerosis is either low or normal. When the pressure is much increased, the presence of a brain tumor should be suspected and drainage would then be a dangerous procedure. On the Neurological Service at the Boston City Hospital, continuous drainage was tried in a number of cases without very satisfactory results. The expected removal of some of the toxic elements by drainage failed to produce any striking improvement in their cases. The treatment has been discontinued after about a year's trial and other methods of treatment have been substituted.

Brickner, at the Neurological Institute in New York, has administered quinine in doses of five to ten grains daily for a period of months, and he feels that it has given relief and has produced a striking remission of symptoms in the early cases treated by him. The same form of treatment is being tried in a number of other clinics at present, but the results are still rather uncertain. It is interesting to note, however, that remissions may take place in multiple sclerosis without or with very little treatment.

In 1930 Sir James Purves-Stewart and Kathleen Chevassut published a paper in which they claimed the discovery of an ultramicroscopic virus as the cause of multiple sclerosis. This "discovery" aroused a great deal of enthusiasm in medical circles. Everybody felt that the problem of multiple sclerosis had been solved finally. Unfortunately it turned out to be a hoax on the part of Miss Chevassut, and Sir James Purves-Stewart has later retracted what he wrote about the virus as the cause: he apologized and stated that he was misled by Miss Chevassut who did most of the work. And so at present we are just as much in the dark about the etiology as we were three years ago. A vaccine also made by Miss Chevassut was used in the treatment of a number of cases but according to Dr. Walshe, an English neurologist, the treated cases were much worse off following treatment.

Dr. Paul deNicola: I have nothing else to say except to call your attention to the relative frequency of multiple sclerosis. I think it is more common than we realize because many of the cases are undiagnosed; they are just not recognizable. These constitute many of the cases in the early stages, of course.

MISCELLANY

ANNUAL MEETING

The next Annual Meeting of the New Hampshire Medical Society will be held at Manchester, May 15 and 16, 1934.

ROCKINGHAM COUNTY SOCIETY

The following officers were recently elected:

President, Herbert L. Taylor, Portsmouth.

Vice President, Donald W. Leonard, Exeter.

Secretary-Treasurer, Wendell P. Clare, Portsmouth.

Delegate, Cleon W. Colby, Exeter.

Medical Jurisprudence Committee, Thomas W. Luce, Portsmouth.

DEATH

KELSO — Dr. Walter Lamson Kelso of Hillsboro died September 9. He was born in 1872 and graduated from the University of Vermont Medical College in 1905.

NEW MEMBER

Dr. James Sanders, Rye, N. H. Graduate of Tufts College Medical School 1932. Licensed 1933.

NICOTIN POISONING FROM ROSE SPRAYS

BY CHARLES D. HOWARD

The average person who professes to know anything about poisons is inclined to assume that hydrocyanic acid, as contained in various cyanides, is the most deadly and most rapidly acting chemical agent of destruction existent. Toxicologists, even, have been known to testify to this effect.

Actually this is not so. Rapidly fatal though hydrocyanic acid is, as a claimant for this honor it has an able-bodied rival in the alkaloid derived from tobacco and known as nicotin. In view of the universal indulgence in the practice of smoking, this may seem like an anomalous statement. The fact that but minute traces of nicotin are absorbed in the practice and that these traces are rapidly eliminated serves, with the principle of habituation, to explain how such a habit is possible.

Yet it is recognized that the ingestion of as little as one grain of nicotin alkaloid is likely to have fatal consequences, and that death therefrom is always prompt and may occur within a very few moments. Though the latter may not be quite instantaneous—as the popular idea about certain potent poisons runs—the period of consciousness and of the ability to perform voluntary acts is likely to be exceedingly brief. In point of fatal dose and rapidity of action, nicotin is fully as poisonous as is hydrocyanic acid.

This matter finds popular interest and instructive application in the fact that nicotin and its compounds are now extensively employed for the destruction of insects. Incidentally, it is highly regrettable that in our agricultural economy the liberal and more or less indiscriminate spraying about of such violent poisons as Paris green, arsenate of lead and nicotin compounds should be necessary for combating insect pests.

Yet these are articles obtainable at any hardware store, with no questions asked. Similarly, although the average druggist is, rightly, exceedingly cautious about dispensing a small quantity of cyanide to an individual, and some members of this profession refuse to sell it at all, it can rather readily be obtained in pound cans by the farmer who wishes it to destroy woodchucks. Professional insect exterminators also use this poison freely as the basis of a fumigant, although the employment for this purpose of the deadly gas resulting is something never to be undertaken by the novice but only by those duly qualified and licensed.

To return to the subject of nicotin, this substance has long been used (frequently under the name of "Black Leaf 40") as a spray for destroying rosebugs, for which it is very effective. Recently it has been proposed for the extermination of bedbugs and of similar vermin, for which purpose it should be well suited.

The danger attending the handling of nicotin preparations and the need of extreme care in this respect have been demonstrated by two fatalities by this poison occurring in this State in recent years.

In both these cases the moral to be drawn is that the keeping on hand of such deadly compounds in containers not conspicuously labeled to show the poisonous character of the contents is the height of carelessness. To the extent that the latter is probably in most cases unwitting, the public needs to be forewarned concerning the exceedingly poisonous character of nicotin.—*Bulletin New Hampshire State Board of Health.*

THE VALUE OF COD LIVER OIL IN THE TREATMENT OF ANEMIA*

BY ARTHUR D. HOLMES, PH.D.,† MADELEINE G. PIGOTT†, AND LAWRENCE P. BOWSER†

IT has been frequently demonstrated that cod liver oil is particularly efficacious in the prevention and cure of certain forms of animal and human malnutrition. Cod liver oil is recognized as being one of the richest naturally occurring sources of Vitamins A and D. Investigations in this laboratory have shown cod liver oil to be a valuable agent for stimulating good growth and bone development in experimental animals[1], and for reducing industrial absenteeism in underweight adults[2]. As a consequence of these observations it was naturally questioned whether cod liver oil was of value in promoting blood regeneration in secondary anemia. A review of the literature revealed a paucity of information in this regard. Koessler, Maurer, and Loughlin[3], who studied the relation of primary and secondary anemia to Vitamin A deficiency concluded that blood regeneration cannot take place without Vitamin A, that the rate and intensity of the blood regeneration is a function of the quantity of Vitamin A, and that a definite relationship exists between a state of chronic vitamin deficiency and certain anemias. In a later paper Koessler and Maurer[4] report that the fat soluble vitamins are factors of primary importance in blood regeneration, that Vitamin A causes a rapid formation of new blood cells, and they recommend supplying Vitamin A to anemia patients by administering 20 cc. of cod liver oil daily. On the other hand Cartland and Koch[5] found that rats maintained for weeks on diets deficient in Vitamin A do not become anemic and they feel that Vitamin A is not specifically essential for the hemoglobin forming process in the rat. To obtain information regarding the supplementary effect of common constituents of basal rations, Krauss[6] added cod liver oil to exclusive whole milk diets fed to rats. He found that cod liver oil did not delay the onset of anemia. In a study of the effect of avitaminosis on hematopoietic function, Sure, Kik, and Walker[7] found in the preophthalmic stage of their animals a suggestion of anemia characterized by reduction in either hemoglobin or erythrocytes. After a more extensive study[8] these authors report that they found no noteworthy disturbance in hematopoietic function during various stages of Vitamin A deficiency as characterized by the severity of ophthalmia and loss of body weight, nor any change in the differential leucocyte count during the onset and remission produced by vitamin therapy. In view of the limited data available and the lack of agreement between investigators who have given this subject attention it seemed desirable to conduct a study of the hematopoietic value of cod liver oil.

Recently weaned albino rats were placed on a diet limited to milk and distilled water. In earlier studies it was found that liquid milk was not entirely satisfactory for experiments of this character since it was necessary to feed the milk to the animals in open dishes, thus the milk became contaminated by the animals and tended to sour quickly, particularly in warm weather. Accordingly it has been the practice in this laboratory to use powdered instead of liquid whole milk. Powdered whole milk (Klim) can be obtained in quantity with a known low copper content and thus it is possible to feed all experimental animals milk of uniform composition. These observations concerning the superiority of powdered milk over liquid milk for the production of experimental anemia in rats are in accord with results reported by various investigators[9].

The experimental animals were housed in a separate room in individual wire cages supported on perforated iron trays as shown in Figure 1. Both cages and trays had been recently galvanized. Groups of 20 to 40 animals were started at frequent intervals on the anemia-producing diet. The animals were fed daily and weighed three times a week.

FIG. I. Laboratory for blood studies.

Blood samples for hemoglobin determinations were obtained by puncturing the rat's tail, near the tip, with a spring blood lancet. This procedure yielded sufficient blood for the test but left no exposed raw tissue as is the case when the end of the rat's tail is clipped. The blood was drawn into a 0.02 cc. blood pipette and quickly transferred to a graduated tube. The

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hemoglobin was determined by a modified Sahli Method, using Osgood-Haskins new permanent standard[10], and Sahli-Haskins tubes, a 100% reading on the tube being equivalent to 13.8 grams of hemoglobin per 100 cc. of blood (manufacturers claim). In general hemoglobin determinations were made at weekly intervals but, as the animals approached the end of the pre-experimental period, hemoglobin determinations were made two or three times weekly.

Obviously the rate of decrease of hemoglobin content of the blood was not the same for all the animals. Hence when the hemoglobin content had been reduced to between four and five grams per 100 cc. of blood the animals were assembled in groups of seven animals each. All of the experimental groups of animals were continued under the same laboratory régime and received the same basal ration of whole milk powder and distilled water. During the experimental three weeks' period the basal ration was supplemented with varying amounts of iron, copper, and cod liver oil.

In order to provide a standard for judging the value of cod liver oil for increasing the hemoglobin content of the blood of anemic animals, it was essential to select supplements which had been demonstrated to be efficient for promoting blood regeneration. The investigations of Mitchell and Schmidt[11], Titus, Cave and Hughes[12], Myers and Beard[13], Drabkin and Waggoner[14] and others indicate that various salts of iron will increase the hemoglobin content of the blood of anemic animals. On the other hand Hart, Steenbock, Elvehjem and Waddell[15]; Hart, Steenbock, Waddell and Elvehjem[16]; McHargue, Healy and Hill[17]; Titus, Cave and Hughes[12]; Underhill, Orten and Lewis[18]; Mitchell and Miller[19]; Keil and Nelson[20] and others have shown that hemoglobin regeneration in anemic rats is more pronounced when iron and copper are used as supplements to the basal anemia-producing ration. Also Schiff, Eliasberg and Joffe[21] believe that copper is of value in the treatment of anemia in nurslings and Josephs[22] has reported more rapid recovery in the treatment of anemic infants with iron and copper than when iron alone was used. On the other hand Gorter, Grendel and Weyers[23] found that the copper content of blood was 1.4 mg. per liter for normal children, 1.0 mg. for newborn infants, 2.1 mg. for children with infectious diseases and 2.1 mg. for anemic children. If these results are of general application it would seem questionable whether copper was required in the treatment of anemia of infancy and childhood. Nevertheless for the purpose of this investigation it seemed desirable to compare the value of cod liver oil for regenerating blood of anemic rats with the results obtained when both iron and copper were used as supplements under comparable conditions. All groups of animals receiving the mineral supplements were fed 0.05 mg. of copper daily since Hart and coworkers[16] have shown this amount of copper administered with iron to be adequate for blood regeneration. The amount of iron fed varied with the different groups.

The iron was fed to the experimental animals as ferric ammonium citrate (Merck's U.S.P.)

TABLE I

THE HEMATOPOIETIC VALUE OF COD LIVER OIL

Group	No. of Rats	Materials Fed	Hemoglobin Start Grams	Hemoglobin End Grams	Body Weight Start Grams	Body Weight End Grams
1	7	0.5 mg. Fe + .05 mg. Cu + 120 mg. CLO	4.53	14.2	142	205
2	7	0.5 mg. Fe + .05 mg. Cu + 60 mg. CLO	4.83	13.8	135	186
3	7	0.5 mg. Fe + .05 mg. Cu + No CLO	4.60	12.1	119	174
4	7	1.5 mg. Fe + .05 mg. Cu + 120 mg. CLO	4.71	14.3	133	193
5	7	1.5 mg. Fe + .05 mg. Cu + 60 mg. CLO	4.67	14.6	133	219
6	7	1.5 mg. Fe + .05 mg. Cu + No CLO	4.74	14.0	121	184
7	7	2.5 mg. Fe + .05 mg. Cu + 120 mg. CLO	4.68	14.3	137	219
8	7	2.5 mg. Fe + .05 mg. Cu + 60 mg. CLO	4.98	14.1	158	212
9	7	2.5 mg. Fe + .05 mg. Cu + No CLO	5.00	14.2	132	199
10	7	3.0 mg. Fe + .05 mg. Cu + 120 mg. CLO	5.02	14.5	135	176
11	7	3.0 mg. Fe + .05 mg. Cu + 60 mg. CLO	4.63	14.7	133	199
12	7	3.0 mg. Fe + .05 mg. Cu + No CLO	4.78	14.6	135	198
13	7	0.5 mg. Fe + .05 mg. Cu + 120 mg. PO	4.91	13.6	145	205
14	7	0.5 mg. Fe + .05 mg. Cu + 60 mg. PO	4.84	13.1	139	215
15	7	0.5 mg. Fe + .05 mg. Cu + No PO	4.60	12.1	119	174
16	7	1.5 mg. Fe + .05 mg. Cu + 120 mg. PO	3.87	13.7	127	198
17	7	1.5 mg. Fe + .05 mg. Cu + 60 mg. PO	4.94	13.8	118	189
18	7	1.5 mg. Fe + .05 mg. Cu + No PO	4.74	14.0	121	184
19	7	2.5 mg. Fe + .05 mg. Cu + 120 mg. PO	5.01	14.4	142	192
20	7	2.5 mg. Fe + .05 mg. Cu + 60 mg. PO	4.35	14.5	126	177
21	7	2.5 mg. Fe + .05 mg. Cu + No PO	5.00	14.2	132	199
22	7	No Fe + No Cu + 120 mg. CLO	4.43	3.53	121	165
23	7	No Fe + No Cu + 60 mg. CLO	4.49	4.12	123	160
24	7	No Fe + No Cu + No CLO	5.12	4.54	138	167
25	7	0.5 mg. Fe + .05 mg. Cu + No CLO	4.60	12.1	119	174

and the copper as copper sulphate (Merck's Reagent). The cod liver oil was prepared from fresh cod livers and had the following chemical and physical characteristics:

Specific Gravity	0.9220
Refractive Index	1.4790
Saponification Value	186.3
Iodine Number	151.9
Free Fatty Acid	0.435
Unsaponifiable	1.295

Its vitamin potency was more than 1000 Vitamin A units and 150 Vitamin D units per gram.

Regardless of the amount of ferric ammonium citrate and copper sulphate fed per day the solutions were so prepared that each animal received 1 cc. of the solution. The amount of iron (as ferric ammonium citrate) fed to the different groups of animals varied from 0.5 mg. to 3.0 mg. per rat daily. Each level of iron was supplemented by 0.05 mg. of copper (as copper sulphate) and by 120 mg., 60 mg. and no cod liver oil.

The complete data collected for the 175 animals used in this study are too voluminous to be reported here. Accordingly averages have been computed for the body weights and hemoglobin values obtained for the seven animals of each experimental group. Table I contains average values for hemoglobin and body weight for each group of animals at the beginning and end of the experimental period and Charts I and II contain average values, as curves, for the entire preparatory and experimental period. It will be noted on referring to Table I that the increase in the hemoglobin content of the blood of the animals (groups 1 to 3), which received 0.5 mg. of iron and 0.05 mg. of copper daily varied with the amount of cod liver oil fed, being 7.50, 8.97, and 9.67 grams per 100 cc. of blood for the groups receiving no cod liver oil, 60 mg. and 120 mg. of cod liver oil respectively per animal daily. Also the animals which received the largest amount of cod liver oil made the largest gain in body weight. On the basis of these data one might conclude that cod liver oil possessed hematopoietic value. However when the amount of iron was increased to 1.5 mg. per animal per day (groups 4 to 6), the animals which received 60 mg. of cod liver oil (Group 5) showed a larger increase in hemoglobin and body weight than either group 4, which received no cod liver oil or group 6 which received 120 mg. of cod liver oil per day. The same situation also exists in the case of group 11 which received 60 mg. of cod liver oil with 3.0 mg. of iron and 0.05 mg. of copper daily. On the other hand group 7, which received 120 mg. of cod liver oil per day with 2.5 mg. of iron and 0.05 mg. of copper showed a larger increase in hemoglobin and body weight than group 9 which received no cod liver oil or group 8 that received 60 mg. of cod liver oil. In view of these variable results these tests were repeated (groups 13 to 21, Chart II) substituting vitamin-free peanut oil for cod liver oil.

The animals (groups 13 to 15) which were fed 0.5 mg. of iron and 0.05 mg. of copper daily supplemented with peanut oil showed less increase in hemoglobin than when 60 mg. and 120 mg. of cod liver oil were fed daily. However when the amount of iron was increased to 1.5

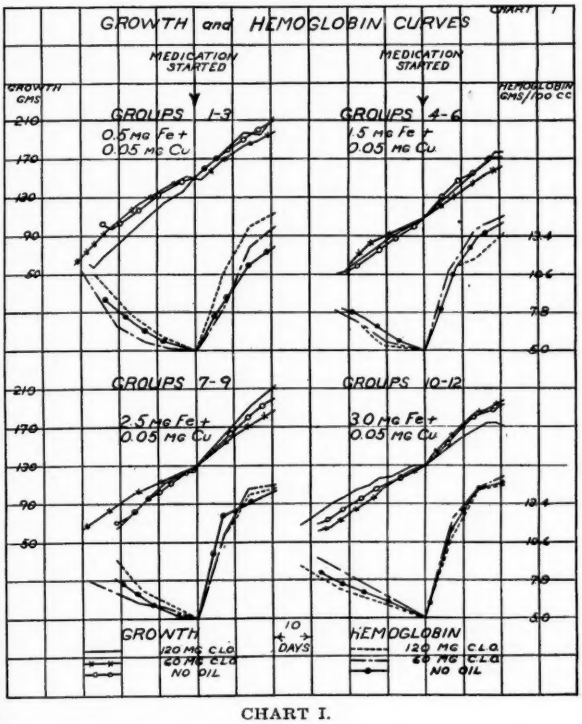

CHART I.

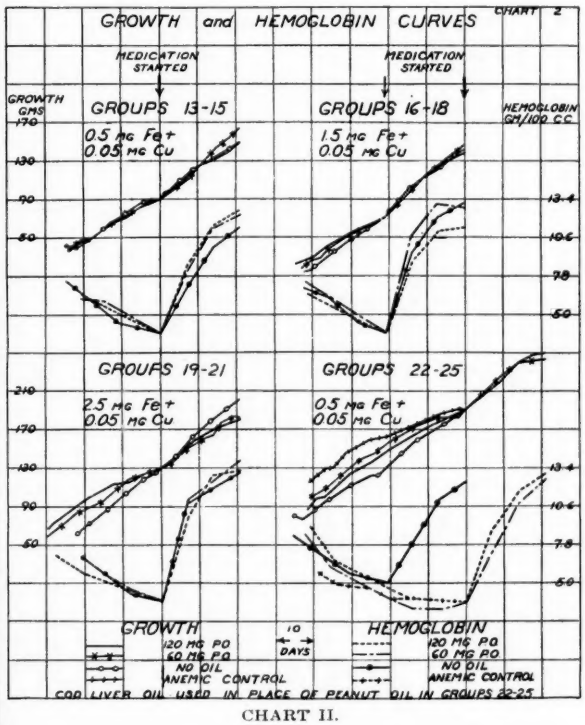

CHART II.

mg. (groups 16 to 18) or to 2.5 mg. (groups 19 to 21) of iron supplemented by peanut oil the increase in hemoglobin content of the blood during the experimental period was comparable to that obtained with equivalent amounts of cod liver oil. From this it is concluded that except when small amounts of iron are adminis-

tered the vitamin-containing cod liver oil possessed no superior blood regenerating value to that of vitamin-free peanut oil.

As a further test four groups of animals (groups 22 to 25) which had developed experimental anemia were given the following treatment during the experimental period: Group 22 received 120 mg. of cod liver oil but no iron or copper; group 23, 60 mg. of cod liver oil per day and no iron or copper; group 24, no cod liver oil, iron or copper; group 25, 0.5 mg. of iron, 0.05 mg. of copper and no cod liver oil. Groups 22, 23 and 24 continued to grow at approximately the same rate but all three groups showed a decline in hemoglobin content of their blood. The seven animals receiving 120 mg. per day and the seven animals receiving no cod liver oil showed the same hemoglobin decline. During the same three weeks the hemoglobin content of group 25 which received the iron and copper increased from 4.60 grams to 12.1 grams per 100 cc. of blood. At the end of the usual three weeks' test period group 24 that had been receiving no iron, copper or cod liver oil through oversight was discarded. Groups 22 and 23 which had been receiving 120 and 60 mg. of cod liver oil respectively were given the same medication as group 25. They showed the same rate of hemoglobin increase as group 25 which received no cod liver oil. From these data it appears that cod liver oil possesses little if any blood regenerating value except possibly when fed in conjunction with small amounts of iron and copper salts.

SUMMARY

A review of the literature revealed confusing reports concerning the value of Vitamin A for promoting blood regeneration in anemia. Accordingly studies were made to determine the hematopoietic value of cod liver oil rich in Vitamin A. One hundred and seventy-five young albino rats made anemic by being confined to a diet of milk powder and distilled water were fed iron and copper supplemented with varying amounts of cod liver oil. Rats which received 0.5 mg. Fe and 0.05 mg. Cu daily supplemented with cod liver oil showed more rapid blood regeneration than rats fed the iron and copper without cod liver oil. However when the amount of iron was increased to 1.5 mg., 2.5 mg., or 3.0 mg. daily there was no significant difference between the rate of hemoglobin increase of rats receiving cod liver oil and that of the controls. Also when Vitamin A free peanut oil was substituted for cod liver oil, essentially the same results were obtained as with cod liver oil. Furthermore when cod liver oil was fed without iron or copper the hemoglobin content of the rats' blood continued to decline. These data apparently indicate that cod liver oil has little if any hematopoietic value.

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MEDICAL PROGRESS

PROGRESS IN THE SURGERY OF THE SYMPATHETIC NERVOUS SYSTEM IN 1932*

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I. General Physiology

IN last year's review[1] Cushing's fundamental discovery that pituitrin or pilocarpine injected directly into the ventricle causes a generalized outburst of activity of the entire cranial portion of the autonomic nervous system was reported. This finding is important from many angles. From the biochemical point of view it demonstrates that the parasympathetic system is controlled by the action of an internal secretion very much as the activity of the sympathetic system is regulated by the hormone adrenalin. From the physiological angle this previously unsuspected action of the posterior pituitary hormone confirms the ideas of other neuro-physiologists—that the diencephalon contains the brain centres of the parasympathetic system. The anatomical work of Popa and Fielding[2] and Basir[3] has indicated a type of "portal circulation" between the pituitary body and the hypothalamic centres, whereby the secretion of the posterior lobe can be carried directly to the parasympathetic ganglia.

Many recent investigations from diverse angles have contributed to our knowledge of these central ganglia of the autonomic system. An excellent summary and correlation of this work has appeared for the first time in this *Journal* during the past year by Fulton[4] and is of such fundamental importance that it deserves to be summarized in considerable detail. To make the story of this research complete we must go back to the work of Goltz forty years ago[5], showing that the decerebrated dog was subject to manifestation of rage accompanied by intense activity of the sympathetic nervous system. Dusser de Barenne[6] made similar observations on cats, and recently Bard[7] has been able to delimit this centre of sympathetic activity to a small triangular segment of the midbrain lying in the sides of the third ventricle and just posterior to the stalk of the pituitary. The investigations of Beattie, Brow, and Long[8] have shown the connections between these posterior hypothalamic nuclei and the lateral horn cells in the thoracic region of the spinal cord, where sympathetic impulses synapse with the cells of the white rami communicantes. Keller[9] has demonstrated that an experimental lesion in the area of the sympathetic centre causes an animal to become sluggish and apathetic, with constricted pupils, low blood pressure, and without capacity to regulate its body temperature.

Whereas electrical stimulation of the above area causes a rise in blood pressure, dilation of the pupils, and cardiac extrasystoles, Karplus and Peczenik[10] and Beattie[11] have noted that stimulation of the hypothalamus two millimeters farther forward in the region of the tuber and supra-optic nuclei causes action currents in the vagus nerves with decrease in heart rate, vasodilatation of the skin and mucous membrane of the stomach, increase in gastric peristalsis and in the secretion of an acid gastric juice. In connection with these last reactions Cushing[12] has called attention to Rokitansky's theory that the cause of peptic ulcer lies in "a diseased innervation of the stomach". Light, Bishop, and Kendall[13] have found that intra-ventricular injections of pilocarpine in rabbits cause gastric peristalsis and such an extreme hyperemia and secretion of gastric juice that, if the reaction is not inhibited by atropine, blood is discharged in the feces and acute ulcers form in the stomach wall.

Another observation in a quite different field lies in a possible localization of a sleep centre in the hypothalamus. Hypersomnia associated with lesions of this region is a well-recognized clinical entity. Hess[14] has recently implanted electrodes here and has been able to reproduce all the signs of normal physiological sleep by weak faradic stimulation. Avertin or tribromethanol is known to act on the subcortical centres and constitutes further evidence for the importance of these centres in the mechanism of sleep.

In summarizing the work of Cannon and others in this field, Fulton states that the sympathetic system, with its center located in the posterior hypothalamus, brings about those adjustments of the body essential for escape and combat. From the viewpoint of the organism as a whole these adjustments are of a spendthrift character, the body temperature is raised, the heart accelerated, and the energy reserves mobilized. On the other hand, activity of the parasympathetic division, with its centre in the supra-optic and tuber nuclei, tends to conserve the resources of the body. From the phylogenetic point of view, it is significant that the hypothalamus, in which these important vegetative centres lie, has changed but little during vertebrate evolution.

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II. Diagnostic Methods

No important new modifications of the methods of blocking the sympathetic ganglia with novocaine for diagnostic purposes have been reported in 1932, but many clinical articles have confirmed the usefulness of these procedures, both in differentiating the spastic and the organic types of peripheral vascular occlusion, and also in studying the pathways of visceral pain. However, an excellent new method of determining the degree of vasospasm in conditions such as Raynaud's disease, thromboangiitis obliterans, and atrophic arthritis has been described. This consists essentially in warming the patient to a point where his heat eliminating mechanism comes into play and a generalized vasodilatation results. Pickering[15] points out that this mechanism is dependent upon the return of warm blood to the vasomotor centre in the brain, and that the central nervous mechanism is sensitive to very small changes in body temperature (.01° to .04° C.).

The method of testing vasodilatation by heating the body was first described by Lewis and Pickering[16], who employed a cabinet heated by electric lights in which the body could be warmed while the hands and feet were exposed to cooler room temperature. Coller and Maddock[17] accomplished the same result by the use of heavy blankets and a rubber sheet. Gibbon and Landis[18] utilized the very simple expedient of immersing the feet or the arms in water heated to 45°C. In all these methods the peripheral skin temperature in normal individuals or in patients with Raynaud's disease rises rapidly to the normal vasodilatation level, whereas in cases with organic arterial occlusion the increase in surface temperature is slow and of slight degree. If the digital temperature rises to above 32°C., organic occlusion of the blood vessels is definitely excluded. If the digital temperature fails to rise, or rising fails to reach 32°C., the presence of organic arterial obstruction is indicated. The simplicity of this test favors its use as a preliminary procedure in the study of cases showing clinical evidence of peripheral vascular disease. The reaction in doubtful cases should be confirmed by the use of one of the methods of novocaine block, general anesthesia, or foreign protein shock. The body heating methods are so simple that they are likely to replace the above tests in the routine study of circulatory cases. They cannot, however, supersede sympathetic novocaine block in the study of visceral pain or abnormal visceral motor innervation.

Johnson[19] has devised an ingenious type of finger plethysmograph in which the changes in volume of the finger with the cardiac pulsations displace a bubble in a capillary tube and the movements of the bubble are photographed by an apparatus similar to that used in recording the electrocardiogram. This gives excellent photographic tracings of the volume of the arterial pulse in the fingers.

III. Operative Methods

There have been no notable papers on the operative technique of sympathetic ganglionectomy, but a number of interesting papers have appeared demonstrating the effectiveness of alcohol injection of these nerves. Reichert[20] reports a series of over 40 cases of alcohol injection in such conditions as Raynaud's disease, scleroderma, and atrophic arthritis with vasomotor instability, thromboangiitis obliterans, and arteriosclerotic claudication. Selection of these cases is made by preliminary paravertebral novocaine injection of the sympathetic chain, which enables the patient to be ambulatory and to assist the physician in judging whether permanent interruption would be beneficial. In such cases where the peripheral temperature was elevated or pain on walking relieved, alcohol injection was usually successful in causing striking improvement. Abbott[21] reports a number of cases of atypical neuralgia, amputation stump and visceral pain which were satisfactorily relieved by similar methods. The risk of this procedure is practically nil.

IV. Effects of Sympathectomy on the Circulation

A. *Anatomy and Physiology of the Vasomotor Nerves.*

Woollard and Phillips[22] report a very thorough study of the pathway of the sympathetic nerves to the extremities. This confirms previous views that the vasoconstrictor fibres pass to the periphery with the peripheral nerves. Furthermore, they have precisely the same distribution as these nerves, i.e., sympathetic fibres in the median or ulnar nerves are limited to the exact territory that these nerves innervate. The ring finger, for instance, receives sympathetic fibres from the median on its radial side and from the ulnar along its medial border. If there were any important fibres descending the ulnar and radial arteries the vasomotor supply could not conceivably correspond in such an exact way with the peripheral nerves. The effect of cutting off the vasomotor innervation is most marked in the digits and the palm and becomes less marked as one advances proximally along the limb. These authors were unable in their experiments to demonstrate any sympathetic fibres going to the vessels in the muscles, and they therefore infer that these must be few in number. While Woollard[23] from his experiments is opposed to Leriche's theory that vasodilatation can be produced by periarterial sympathectomy, he feels that there is some surgical

evidence that causalgic pain may be relieved by this operation. The problem here that needs solution is how far the sensory fibres run in the blood vessels before they rejoin the peripheral nerves, and although the answer to this question cannot be stated with certainty, it is probable that the distance may be considerable.

Gilding[24] has experimentally confirmed Woollard's observations in an entirely different manner. In anesthetized animals the stellate ganglion on one side was exposed, enclosed in glass shielded electrodes, and stimulated faradically. During stimulation brom-phenol blue was injected rapidly into a vein. Sixty seconds after the beginning of the injection the abdomen was opened and the animal rapidly exsanguinated by cutting the abdominal aorta and inferior vena cava. The vasoconstriction produced was readily manifest by the absence of staining in the ischemic areas, in marked contrast to the heavily stained neighboring tissues. From these experiments Gilding observed: (a) that vasoconstrictor impulses stop short at the midline of the body, while arterial channels cross freely over the midline; (b) that such small areas of skin as that covering half a cat's digit are deprived of sympathetic innervation by section of the local minute sensory nerves, proving that constrictor impulses do not travel even for such small distances in the arterial plexus; and (c) that stimulation of the stellate ganglion causes vasoconstriction in the skin of the corresponding arm, side of the head, mucous membranes, muscles, bone, bone marrow, meninges, choroid plexus, and thyroid gland.

Oughterson, Harvey, and Richter[25] record the postoperative course of a patient following unilateral cervicodorsal and lumbar ganglionectomy. They show, by careful temperature studies five months later, that there has been a definite falling off in the circulation rate of the sympathectomized extremities and that novocaine injection of the peripheral nerves will cause a secondary rise to the full vasodilation level. In a series of experiments on dogs they were able to show that a certain number of vasomotor fibres leave the spinal cord as low as its sixth lumbar nerve root, whereas in the usual form of human operation the fourth lumbar ganglion is the lowest to be removed. They conclude that the not infrequently unsatisfactory results obtained by operative procedures designed to interrupt sympathetic vasomotor pathways may in part be explained by our lack of knowledge of the exact course pursued by these fibres and particularly of the levels at which they leave the spinal cord. Both clinical and experimental evidence point to these pathways leaving the cord over a much wider distribution than that originally described by Gaskell and by Langley.

While there have been frequent reports of unsatisfactory results following removal of the upper thoracic sympathetic ganglia, the results in lumbar ganglionectomy have been pretty uniformly successful. Moore, Williams, and Singleton[26] have shown by x-rays after the injection of radio-opaque media into the abdominal aorta in cats a striking degree of vasodilatation that occurs on the side from which the lumbar ganglia have been removed. They have followed this reaction up to eleven weeks after operation. Section of the sciatic nerve as it leaves the pelvis also causes a widespread vasodilatation, but in no instance has periarterial sympathectomy produced any demonstrable change in the artery. Reichert[27] adds further experimental evidence concerning the ineffectiveness of periarterial sympathectomy in increasing the blood flow to an extremity. In three dogs no increase in temperature could be made out in the operated leg and on exposure in a cold room operated and unoperated extremities showed an equal fall in surface temperature.

Tower and Richter[28] reported last year that the white communicant rami on section regenerate on the average within twenty-eight days. They have since studied the question of regeneration of the postganglionic gray rami by similar methods, utilizing the increase in electrical skin resistance which follows the abolition of the secretory activity of the sweat glands when the sympathetic nerves are paralyzed. They conclude that there is no evidence of regeneration when the postganglionic fibres of the gray rami are divided.

Johnson, Scupham, and Gilbert[29], by using the finger plethysmograph described above[19], conclude that while there is an early marked increase in blood volume entering the finger with each heart beat after sympathectomy, there is a progressive recovery of independent arterial tone, so that the circulation has dropped back to its preoperative level within twenty-one days. This conclusion is unlikely to appear as valid to anyone who has followed a large series of sympathetic operations in man or animals, and is directly opposed to the recent work of Herrick, Essex and Baldes[30]. In approaching this question they measured the blood flow directly in the femoral artery of dogs twelve, nineteen, and thirty-four months after lumbar ganglionectomy. In every instance the flow in the femoral artery on the operated side was at least double that of the control side. They concluded that the increase in blood flow following lumbar sympathectomy was apparently permanent.

B. *Effect of Sympathectomy in Raynaud's Disease.*

Allen and Brown[31] have given a very thorough review of their experiences in a series of 204 cases of Raynaud's disease at the Mayo Clinic. These were subdivided into: (1) un-

complicated type, 147 cases, (2) patients with superimposed scleroderma or arthritis, 51 cases, and (3) types of trophic changes or recurrent infections unassociated with scleroderma or arthritis, 60 cases. They sum up the requirements for a diagnosis of Raynaud's disease as: (1) intermittent attacks of discoloration of the extremities, (2) symmetrical or bilateral involvement, (3) absence of clinical evidence of occlusion of the peripheral arteries, (4) gangrene or trophic changes limited in large degree to the skin, (5) two years the minimal period of duration, (6) absence of organic disease to which the vasomotor changes might be secondary, and (7) predilection of the disease for females. Their study of these cases indicated that the uncomplicated type of the disease is an equivalent of a psychoneurosis or neurasthenia, and that operation on the sympathetic nervous system is the best method of treatment, offering a cure in the uncomplicated types of the disease.

Allen[32] and White[33] have reported a similar, smaller series of cases from the Peripheral Vascular Clinic of the Massachusetts General Hospital. In these cases, prior to 1932, the first and second thoracic ganglia were removed for the relief of Raynaud's disease in the arm and the second to fourth lumbar ganglia when the disease involved the lower extremity. In a series of 21 cases the lumbar operation was uniformly successful, but in 14 out of 23 operations in the upper extremity the symptoms of vasomotor spasm recurred at the end of six to eight months. In searching for the reason for these recurrences it was obvious that sweating and other signs of sympathetic activity had slowly recurred, so that although the immediate effect was uniformly successful, the end results were far from satisfactory. Since the time of this report the inferior cervical ganglion has been routinely removed as well as the first and second thoracic. With this modification in technique there has been no recurrence of vasomotor activity. As a result the failures are fewer, but nevertheless there are still certain instances of failure which are now being further investigated. Spurling, Jelsma, and Rogers[34] report similar recurrences in two out of three advanced cases of Raynaud's disease treated by sympathetic ganglionectomy. In one case all the digits of one hand have been lost by dry gangrene after the removal of the entire sympathetic chain from the level of the superior cervical to the third thoracic ganglion, as well as periarterial sympathectomy of the axillary artery. Histological studies on the amputated digits show sclerotic and hypertrophic changes in the intima and media of the larger arteries, causing practically complete occlusion of the lumen.

Numerous histological examinations of the sympathetic ganglia in Raynaud's disease have shown no outstanding constant abnormality. Gagel and Watts[35], however, report interesting findings from an autopsy in Foerster's clinic showing definite pathology in the lateral horn cells. Clark's column is known to contain the motor cells of the sympathetic fibres which leave the cord in its thoracicolumbar segments over the anterior roots to enter the white communicant rami. In this case nearly all of these cells showed degenerative changes which were not present in the nerve cells of the anterior and posterior horns.

C. *Results of Sympathectomy in Thromboangiitis Obliterans.*

Adson and Brown[36] review the experience at the Mayo Clinic in a series of 100 consecutive cases of Buerger's disease. Ninety-six of these patients were male and four were female. There were 85 bilateral lumbar ganglionectomies and 15 bilateral cervico-thoracic operations. During the early development of sympathectomy in thromboangiitis obliterans approximately one patient in seven was sympathectomized, but during the past two years the ratio has increased to one in three. These patients are given intravenous typhoid vaccine or a regional or spinal anesthetic with novocaine to determine the degree of vasospasm. In all instances in which there is a rise in skin temperature of the digits twice as great as the rise in mouth temperature during the height of the fever, or a rise of 3°C. after novocaine block, they believe operation is indicated. They feel that the operation should always be done bilaterally, as even when only one extremity is diseased the other is pretty sure to be involved at a later date. The number of amputations necessitated in this series has been markedly decreased. The operation in cases of thromboangiitis obliterans is of no value for neuritic pain, and carries a mortality rate of about five per cent.

Allen[32], in describing the handling of similar cases at the Massachusetts General Hospital, points out that many of these cases can be treated equally well by simple peripheral neurectomies in the lower leg under local anesthesia. The mortality in these cases is nil, neuritic pain is completely relieved, and circulation is improved for several months just as much as after the more extensive operation of ganglionectomy. While this increase in circulation disappears as the nerves regenerate, it usually lasts long enough to tide the patient over an acute crisis of arterial occlusion until his collateral circulation has had a chance to become established.

Maddock and Coller[37] have demonstrated that there is a distinct peripheral vasoconstriction from the active products absorbed from tobacco smoke. Skin temperature readings showed a fall of from 2° to 6°C. while smoking cigarettes, with a gradual return to normal temperature on cessation of smoking. Whether this is due to local changes in tissues from the absorbed nico-

tine or a direct effect on the sympathetic nervous system remains as yet unknown.

D. *Results of Sympathectomy in Other Conditions of the Extremities.*

Kotzareff and also Braeucker reported prior to 1929 the use of sympathectomy for the relief of excessive sweating in the extremities. Adson and Brown[38] report a recent similar successful case.

Key and Moore[39] have tested the effect of sympathectomy on the healing rate of bone and articular cartilage in cats. They find no difference in the rate of repair between the sympathectomized and the control extremities.

In a clinical study of traumatic osteoporosis, Fontaine and Herrmann[40] summarize their experience of 22 cases treated by sympathectomy. When untreated, the resulting pain, immobility, and extreme decalcification often lead to ankylosis of the involved joint. In cases treated early by sympathectomy, recovery of joint mobility, freedom from pain, and redeposition of calcium in the bones were most satisfactory.

V. Pain

Langley, defining the functions of the autonomic nervous system in 1921, stated: "The autonomic nervous system consists of nerve cells and nerve fibres, by means of which efferent impulses pass to the tissues other than the multinuclear striated muscle." This dictum that the autonomic was purely an efferent system concerned with the motor control of the viscera and not directly concerned with visceral pain has been slowly discarded. The clinical and experimental observations of recent years, which have been reviewed in this journal, have gradually forced neurophysiologists to give up this limited point of view and to admit that there are direct sympathetic pathways which carry pain sensation from the various viscera to the brain. The general recognition of this fundamental principle constitutes one of the outstanding advancements in the general knowledge of this field during the past year.

Davis, Pollock, and Stone report a series of experiments demonstrating the presence of direct pain pathways in the sympathetic nervous system[41]. Davis, with Hart, and Crain[42] reported in 1929 that gall bladder distention in a cat uniformly produced evidence of pain, as shown by struggling, respiratory, and other reflex changes. When the right splanchnic nerve was divided all evidence of pain was abolished. This might be interpreted in one of two ways: (1) that pain pathways run in the sympathetic system to enter the cord directly, or (2) that visceral afferent stimuli set up axone reflexes in the sympathetic ganglia, causing certain obscure changes in the periphery which are referred to the central nervous system as pain by the somatic nerves. In this year's work they have investigated these two possibilities and have shown that when all peripheral sensation is eliminated by sectioning the intercostal nerves just lateral to the inflow of the sympathetic fibres, pain reactions are still present. When either the right splanchnic nerve or the posterior roots of the intercostal nerves are severed, all sensation of pain disappears. This proves in a conclusive fashion that abdominal visceral pain ascends the splanchnic nerve and, after traversing the sympathetic ganglia and their white rami, enters the spinal cord by way of its posterior roots. Although the pain is not abolished by peripheral section of the intercostal nerves, it is somewhat less intense. This may mean that the mechanism of referred pain described by Head and MacKenzie is a second mechanism in visceral pain sensation.

Spiegel[43], who has worked on the centrifugal pathways of the sympathetic system, has shown that the efferent pathways from the viscera lie in close relation to the pyramidal tract. The afferent fibres lie in the spinothalamic tract intermingled with the somatic afferent fibres, but the former frequently cross from one side to the other by short neurones. Therefore unilateral cordotomy cannot be counted on to relieve contralateral visceral pain, as it does so effectively in the case of pain from an extremity. In this observation Spiegel has confirmed the findings of Davis, Hart, and Crain[42] quoted in last year's review.

Davis, Goode, and Weiss[44] have demonstrated that the afferent spinal sympathetic pathway of the vomiting reflex is located in the anterolateral tracts of the spinal cord. It seems, therefore, that visceral and cutaneous pain pathways are closely associated in the posterior roots and in the spinal cord, and that the afferent fibres in the sympathetic system are probably no different from those running in the ordinary peripheral nerves.

Heinbecker[45], after studying the peripheral neurones of the sympathetic nerves, describes three types of fibres. The thinly myelinated and non-myelinated fibres carry motor impulses and have a slow rate of conduction, whereas the large myelinated fibres are afferent, have a rapid conduction rate, and correspond to afferent fibres in the somatic nerves. It is noteworthy that these fibres are found in the vagus and in all the cardiac nerves.

A. *Atypical Neuralgias of the Head.*

From the knowledge that stimulation of the superior cervical sympathetic ganglion produces severe pain in the head, it has been assumed that migraine and certain neuralgias of the head, which have failed to be relieved by section of the posterior root of the Gasserian ganglion, may be transmitted over the cervical sympathetic trunk. Davis and Pollock[46] have studied the possibilities of pain transmission over the cervical sympathetic nerves. They have shown that stimulation of this ganglion

after section of the cervical posterior spinal roots and the posterior root of the trigeminal nerve produces no pain. Furthermore, stimulation of the sympathetic trunk itself below the ganglion fails to produce pain, while section of the trunk below the superior ganglion does not abolish the pain when the ganglion is stimulated. They conclude, therefore, that stimulation of the superior cervical sympathetic ganglion produces an effect that is carried by way of the postganglionic efferent fibres to the structures innervated by these nerves. These efferent impulses produce an effect on the skin and other structures the exact nature of which is unknown. It is possible that a metabolite is liberated, which in turn stimulates the ordinary sensory nerve endings of the fifth cranial and upper cervical somatic nerves. Wilson[47] in a clinical study of atypical neuralgias of the head agrees with Davis and Pollock that the pain is probably caused by a sympathetic disturbance in the face which is translated into pain by the sensory nerves. This type of pain can occasionally be relieved by psychotherapy and, as reported previously by Mixter and White[48], in carefully selected cases by cervicothoracic ganglionectomy.

Helson[49] studied the partial return of sensation which occurs after section of the posterior root of the Gasserian ganglion in cases that had been operated in Frazier's clinic. Sensations of light pressure, pain, and medium range of temperature are permanently lost, but the perception of pressure and extremes of temperature return to a surprising degree. Past evidence points to the fact that there are a few sensory fibres in the facial nerve which may carry pressure sensation. Residual perception of heat and cold is probably transmitted by the sympathetic. Helson's findings support the conclusions of Foerster, Altenburger, and Kroll that the sympathetic system plays a sensory rôle in every part of the body, and that to deafferent the face completely, the sympathetic supply must be taken away as well as the fifth cranial nerve. In spite of the disagreement of this report with the experimental findings of Davis and Pollock, the results of sympathectomy in cases of neuralgias of the head which have persisted in spite of section of the fifth cranial root, give clinical support to Helson's observations.

B. *Cardiac Pain.*

Leriche and Fontaine[50] at the forty-first congress of the French Surgical Association gave a very thorough report of the surgery of angina pectoris. In this they report current investigations on the coronary arteries and a complete review of the world literature on resection of the cervical sympathetic trunks for the relief of cardiac pain. In 46 cases of cervical sympathectomy without removal of the stellate ganglion, 58 per cent experienced satisfactory relief. When the stellate ganglion also was removed, the good results increased to 70 per cent. The authors report 11 cases of their own in detail. Levy and Moore[51] have reviewed 57 cases of paravertebral alcohol injection in the American literature, and add nine new cases of their own. Eighty-five per cent of these cases showed satisfactory improvement with only 15 per cent of failures and no fatalities. The only objections to alcohol injection therapy recorded to date are (1) an occasional failure to destroy successfully all the upper thoracic ganglia, and (2) the resultant intercostal neuritis which is a temporary annoyance in nearly every case. From our present knowledge of cardiac innervation, it appears justifiable to conclude that cardiac pain can be relieved in every case where the upper thoracic ganglia are completely destroyed.

Adson[52] feels that cases to be subjected to surgical treatment for angina pectoris should be divided into two groups. Superior cervical ganglionectomy should be used on the younger group if they present vasomotor phenomena and if they would otherwise be compelled to undergo medical treatment for years. Paravertebral alcohol injection of the upper thoracic nerves is the procedure of choice in senile patients when medical treatment fails and when the lesion is due to sclerotic changes rather than to vasomotor spasm.

C. *Abdominal Pain.*

Alvarez[53], who has made a long study of the nerve supply of the abdominal viscera, concludes that the vagus nerves carry so few sensory fibres that they can be ignored whenever efforts are being made to relieve abdominal pain. That these fibres are unimportant is shown by the fact that peritonitis is painless in patients who have suffered injury to the upper thoracic portion of the spinal cord, and by the fact that abdominal operations can be done painlessly under splanchnic blocking or spinal anesthesia. The phrenic nerves carry a few sensory fibres which are responsible for the shoulder pain in gall-stone colic or subdiaphragmatic inflammation. In studying cases of abdominal pain at the Mayo Clinic, spinal anesthesia is first induced and then observations are made to determine the level to which cutaneous anesthesia reaches at the time that the visceral pain is relieved. In this way the highest segment from which pain-carrying fibres pass to the cord can be identified. Later paravertebral novocaine injections, as described by Kappis, by Läwen, and by Preiss, can be used to determine the exact segments. When in a given case it can be shown repeatedly that blockage of the nerves at a certain level will stop the pain, the patient can be fairly certain of securing permanent relief from section of the corresponding posterior roots, or by alcohol injection or resection of the sympathetic rami.

D. *Pelvic Visceral Pain.*

Elaut[54], and also Fontaine and Herrmann[55] have reviewed the anatomy and physiology of the nerve supply of the uterus and ovaries. They claim that simple removal of the so-called pre-sacral nerve or superior hypogastric plexus (the operation of Cotte) is sufficient to relieve the pain of intractable dysmenorrhea. Fontaine and Herrmann review the results in 22 such cases treated in Leriche's clinic in Strasbourg. In 15 followed for periods of over four months there were only two failures and 13 completely successful results. They further recommend that the excruciating pain associated with inoperable pelvic neoplasms should be treated by this operation with the addition of resection of the third and fourth lumbar ganglia on each side. In all cases, even those in which the most extensive pelvic sympathectomy was performed, there have never been any serious complications that could be attributed to this procedure: no motor paralysis, no ascending urinary tract infection due to bladder paralysis, and no sensory disturbances of the skin. Since this operation gives the surgeon an opportunity of verifying the extent and nature of the neoplasm, the authors recommend that it be used in preference to cordotomy in any malignant condition which is found to be localized in the pelvic viscera. The above claim is more optimistic than Learmonth's report of last year, in that he felt that a considerable portion of the pain in malignant disease of the pelvic viscera is transmitted to the inferior hypogastric plexus and enters the spinal cord by the white rami of the second, third, and fourth sacral nerves. When these structures are removed, complete paralysis of the bladder results. My personal feeling is that it is best to try the resection of the presacral nerve along with the lower lumbar sympathetic ganglia in explorations for advanced pelvic malignancy, reserving cordotomy for the occasional cases which fail to secure sufficient relief.

Wharton[56] has studied the nerve supply of the ureter. In ordinary dissections one can make little out of the finer sympathetic plexuses, but by block removal and the use of Siehler's staining technic, in which the nerves are stained while other tissues remain in a soft and pliable state, Wharton has made some excellent anatomical preparations. These show that the ureters receive a nerve supply which is independent of that of the kidneys and bladder. The sympathetic grey rami run directly to the ureter from (1) the lowest renal ganglion at the upper end of the spermatic plexus, (2) the abdominal sympathetics—lumbar ganglia, pre-aortic, and presacral plexuses. The ureter can easily be denervated at operation by lifting it from its bed and sectioning all the nerve filaments which join it. From the physiological point of view, clinical observations have shown that cutting these nerves does not interfere with the motor function of the ureter and does not cause atony, strictures, or hydroureter. In two cases of chronic ureteral colic without other disease the application of these principles has resulted in complete and lasting relief.

VI. Visceral Motor Function

Whether megacolon is due to some abnormality of the colon itself or to its extrinsic autonomic nerve supply has been an open question. Adamson and Aird[57] point out that it is frequently associated with megalo-ureter or dilated bladder and that the only factor which is shared in common by the urinary tract and the colon is their nerve supply. These investigators have also produced megacolon experimentally in cats after resecting the nervi erigentes. In this operation they removed the parasympathetic rami of the sacral nerves, fibres whose function is to relax the internal sphincter and increase the muscular tone of the lower colon. The sympathetic inhibiting innervation via the preaortic and presacral plexuses was thus left intact. Visible enlargement of the colon was observed in eight weeks and was gradually progressive as long as the animals lived.

In writing an annual review of progress of knowledge in a new subject such as the autonomic nervous system, it is interesting to note the phases of enthusiasm which follow in the wake of any new procedure, but which disappear from the literature when they fail to yield lasting satisfactory results. Such has been the course of periarterial sympathectomy for the improvement of circulation in the extremities and of sympathectomy in spastic conditions. No important articles on these subjects have appeared during the past year, in the American or British medical journals at least, except to condemn these operations as unsound. Sympathectomy in the other conditions reported above, such as the peripheral vascular disturbances associated with abnormal tone of the arteries, visceral pain, and abnormal visceral motor function, are now emerging from the experimental phase to a stage where their limitations are recognized and their results can be counted on.

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CASE RECORDS
of the
MASSACHUSETTS GENERAL HOSPITAL

ANTE MORTEM AND POST MORTEM RECORDS AS USED IN WEEKLY CLINICAL-PATHOLOGIC EXERCISES

EDITED BY RICHARD C. CABOT, M.D.
F. M. PAINTER, A.B., ASSISTANT EDITOR

CASE 19431

TEN MONTHS' DYSPNEA IN A MAN OF FORTY-TWO

MEDICAL DEPARTMENT

An Irish-American shoe factory operative forty-two years old entered through the Emergency Ward complaining of progressive shortness of breath of ten months' duration. The history he gave was only moderately consistent.

The onset was gradual and was accompanied by dull constant pain over the upper chest and back radiating down both arms, a sense of pressure across the chest and productive cough. The pain soon disappeared. The cough persisted. The dyspnea gradually grew worse. He slept poorly, needing at least two pillows. For two months he had had evening edema of the ankles. For five weeks he had led a bed and chair existence, with marked orthopnea. For two weeks he had had swelling of the scrotum.

X-ray examination in the Outpatient Department seven months before admission to the wards showed general enlargement of the heart, greatest to the left; slight increase in the supracardiac shadow; no definite evidence of dilatation. The lung fields were clear.

One brother had a weak heart. The patient's wife had one miscarriage and died in childbirth.

The past history is negative except for a penile sore nineteen years before admission and some shooting pains seven years before admission.

Physical examination showed a moderately well nourished man sitting up in bed breathing with slight effort. The lips were slightly cyanotic. The neck veins were full. The sclerae were slightly icteric. The lungs showed moist râles at both bases in front and at the left base behind; at the right back dullness to flatness below the angle of the scapula with distant breath sounds and decreased tactile fremitus. The heart was enlarged to the left, the left border of dullness 11 centimeters from midsternum, one centimeter outside the midclavicular line, the right border of dullness 3 centimeters, the supracardiac dullness 6. The action was rapid, the rhythm grossly irregular. There was a diastolic murmur in the third left interspace directly following the second sound; a late diastolic at the apex. No thrill was made out. The pulses were equal and synchronous. The blood pressure was 135/40. The artery walls were normal. The abdomen appeared full. There was slight dullness in both flanks and questionable shifting dullness. No wave was made out. The liver edge was felt four fingerbreadths below the costal margin. The tip of the spleen was just palpable. There was massive edema of the scrotum and penis, gross pitting edema of the feet and legs.

The urine is not recorded. The blood was normal. A Hinton test was weakly positive.

The temperature was 97.4° to 97.9°, the pulse 100 to 102. The respirations were normal.

The morning after admission the patient appeared more ill and had Cheyne-Stokes respiration. No diastolic murmur was heard at the apex. Digalen was given, six ampoules intravenously. Almost immediately afterwards the patient vomited, the heart stopped and the lungs appeared to be full of moisture.

CLINICAL DISCUSSION

DR. RICHARD C. CABOT: "A man forty-two years old, complaining of progressive shortness of breath of ten months' duration." As soon as we read that, a certain cause of disease should come into our minds. He is a middle-aged man, rather younger than the period at which hypertension ordinarily shows. He is rather beyond the age when rheumatic heart disease usually shows itself. One begins to guess at the possibility of a syphilitic trouble, because all the treatises tell us that when a middle-aged man, with nothing before to lead up to it, begins to have symptoms of heart trouble syphilis should be sought for.

He had "dull constant pain over the upper chest and back, radiating down both arms, a sense of pressure across the chest and productive cough." That of course we get with hypertension or a syphilitic process either. Pains of this sort disappear ordinarily when compensation fails. It is one of the few good things we can say about angina pectoris that when compensation fails you are apt to get over your pain; you do not have to suffer both at once. We shall be interested to see whether the x-ray films will tell us about syphilis.

DR. AUBREY O. HAMPTON: His heart, according to our measurements, is enlarged, but not so much as usual in a patient with syphilis of the aorta. I assume it was left ventricular. The supracardiac shadow is increased in width, but not markedly. It is seven centimeters in transverse diameter, without much tortuosity of the aorta. We should say it is perhaps one centimeter above normal in the postero-anterior view, and in the oblique view it is only one half centimeter above normal. So that we have not quite enough to make a positive diagnosis. We can only say that the aorta is slightly dilated and there is left ventricular hypertrophy.

DR. CABOT: One of the points that I have often brought up to discuss with Dr. Holmes is as to whether we could get aid from the radiograph in the discrimination between arteriosclerosis and syphilitic aortitis. Up to date I have got no help.

DR. HAMPTON: I think the easier the diagnosis is clinically the less we help in the general run of cases. Of course in aneurysms and dilatation of the aorta without much valve disease we aid considerably. We can also determine whether or not the aorta is dilated in either case.

DR. CABOT: In aneurysm there is nothing that helps so much as x-ray; but it is in these cases of suspected syphilitic aortitis without aneurysm that I, so far, have not had help from x-ray.

His wife had one miscarriage and died in childbirth. That, of course, is not enough to give us any considerable evidence about syphilis. One miscarriage suggests it, but is not enough.

Is anything more said in the record about where those shooting pains were? If you had shooting pains in your head that would not mean much; if in the legs they would mean a good deal.

MISS PAINTER: It is not recorded.

DR. CABOT: If you or I had begun to take that history we should have followed it up and not left it at that.

The "lung findings" may all perfectly well be due to enlarged liver. We seldom can tell whether it is liver or fluid at the right base.

The heart measurements by percussion come very close to the x-ray. The x-ray says ten and five, that is fifteen across; they made it eleven and three, which is only one centimeter out, the supracardiac dullness, six. That is as good as we can expect certainly from percussion.

Here again, when we write a record and are trying to make out whether there is mitral disease or not, we should say something about the pitch of the murmur at the apex. A mitral diastolic murmur is a very low-pitched murmur; it is something like two octaves lower than the aortic diastolic. An aortic diastolic murmur is a high pitched murmur practically always. It is a hissing, blowing sound, not a low rumble. So when we make a diagnosis of a late diastolic at the apex we should put down its pitch. Anybody can tell a rumble from a blow. That is all that is meant by low pitch. A low pitch is a rumble. Dr. Frederick C. Shattuck, who used to be professor of medicine here, could tell those differences of pitch perfectly, yet he had no trace of any musical ear.

"No thrill was made out." Here they are thinking of the differential point that we are after. Nobody would think of a thrill at the base in a case like this. It does not say whether the pulses were irregular or not; we should expect them to be irregular from what has been said. "The blood pressure was 135/40," so we are pretty sure there is aortic regurgitation. There may be mitral disease too. We cannot be sure there is not that also, but the positive evidence is all the other way. He must have died pretty quickly so that they did not have time to record the urine. The Hinton test leaves us uncertain.

It is comforting later to hear that "no diastolic murmur was heard at the apex." That supports our idea that all the trouble is at the aortic valve.

DIFFERENTIAL DIAGNOSIS

Here is a man of forty-two, who up to that age had no cardiac symptoms but who had a penile sore which may have been syphilis and may not, who then had the usual symptoms of a failing heart and died in his first failure of compensation. I do not think I ever saw a case of rheumatic heart disease which met death in the first attack. It makes all the difference in the world in practice if you can say to the patient or his friends, "This patient is very sick, but he will get better because it is his first decompensation and rheumatism is its cause." On the other hand, if you really think it is syphilis you cannot say that. They go along extraordinarily well for a long time, but when they fail they usually die in the first attack. There is a very sharp difference there. Years ago when I gave a postgraduate course here we used to assemble groups of patients to illustrate the different types of disease. We took their names from the catalogue and did not know whether the patients were alive or not, but it was a striking thing that the mitral cases came in year after year; most of the aortic cases were dead. We noted the difference though we did not know enough to know that they were due to syphilis. That was all prior to the working out of what we depend so much on in diagnosis now, I mean the knowledge of etiology in cardiac disease.

Here we have no rheumatic history. That does not prove it was not a rheumatic heart. Many cases believed by the pathologist to be rheumatic have no rheumatic history. We have a weakly positive Hinton test, and the Hinton is the most delicate test we have. That does not prove it is not syphilis. I wish we knew more about those shooting pains, but we never shall. The fact that he had so much pain across the chest without any hypertension, and at forty-two, prejudices me further in favor of syphilis. So it seems to me on the evidence we have we should say that this aortic regurgitation is due to syphilitic aortitis, aortic regurgitation with enlargement of the heart, especially of the left ventricle, and essentially nothing else.

A STUDENT: Is there any significance in the

shadow at the conjunction of the sternum and the manubrium?

DR. HAMPTON: The film is rotated so that the junction is out beyond the edge of the mediastinum. This is the manubrium and this is the trachea.

DR. CABOT: As we come to the autopsy it seems to me that there are certain things that are certain. We are sure of aortic regurgitation. We are sure of hypertrophy and dilatation of the heart. Beyond that the case is uncertain, but the weight of evidence seems to point to syphilis.

A STUDENT: Have we any record of his blood pressure when he was in the Outpatient Department seven months ago?

DR. CABOT: That is a good point, because it might have been high then and have fallen since.

A STUDENT: Because he had such a large pulse pressure.

MISS PAINTER: The blood pressure in the Outpatient Department was 125/70.

DR. CABOT: That is not so large a pulse pressure as we should expect with aortic regurgitation. The systolic pressure is normal.

DR. TRACY B. MALLORY: It was felt also by the man who examined him that he had auricular fibrillation. I think there was no electrocardiogram, but they felt quite certain.

DR. CABOT: Auricular fibrillation is of course commoner in rheumatic disease than it is in syphilitic. It occurs in all types of heart disease. I have never heard of any type which may not have auricular fibrillation at the end. But it is certainly commoner in the early stages of rheumatic disease than of the other types. I do not think the presence or absence of fibrillation helps much in the late diagnosis; but if he had it in the beginning of his trouble, then it would be a piece of evidence pointing towards rheumatic disease.

CLINICAL DIAGNOSES (FROM HOSPITAL RECORD)

Luetic heart disease.
Auricular fibrillation.
Congestive heart failure.

DR. RICHARD C. CABOT'S DIAGNOSES

Syphilitic aortitis.
Aortic regurgitation.
Cardiac hypertrophy and dilatation, especially of the left ventricle.
Chronic passive congestion.

ANATOMIC DIAGNOSES

1. *Primary disease.*
   Syphilitic heart disease with aortic insufficiency.
   Syphilitic aortitis.
   Cardiac hypertrophy and dilatation.
   Congestive heart failure.
2. *Secondary or terminal lesions.*
   Pulmonary congestion.
   Hydrothorax, bilateral.
   Ascites.
3. *Historic landmarks.*
   Prostatic hyperplasia, slight.

PATHOLOGIC DISCUSSION

DR. MALLORY: We found a somewhat—only moderately—enlarged heart weighing 425 grams. The average case of aortic insufficiency from lues at the time it comes to autopsy is apt to give a very big heart, which may on occasion run up over a kilogram in weight. So I should say this was distinctly a small heart to find at the time of death from syphilitic heart disease.

The aortic valve measured 7.4 centimeters in circumference. In other words, the ring was not dilated at all but the cusps were very definitely separated, two to three millimeters in each of the commissures, which is a characteristic luetic lesion. The mouth of the left coronary was slightly narrowed, as is so common in lues, but the remainder of the coronaries once we got beyond the mouth was perfectly good, as is usually the case. There was no acute endocarditis, but we noticed on the surface of the interventricular septum—the surface pointing towards the left ventricle—a white opaque spot. In cutting down into that we found it projected into the myocardium for a little over a millimeter,—not very much, but it seemed to us a very unusual finding. There was another similar spot in one of the papillary muscles. The aorta showed slight but quite typical luetic scarring, without any marked degree of dilatation.

The other organs were essentially negative except for chronic passive congestion and anasarca.

Microscopic examination confirms beyond any doubt the luetic character of the aortitis, and shows a quite definite acute myocarditis with foci of degenerating muscle cells and a certain amount of polynuclear infiltration.

The question comes up whether that is really a luetic myocarditis. I am rather tempted to believe it is. We have not yet done a spirochete stain on it. That would prove it if it were positive, and still would not rule it out if it were negative.

DR. CABOT: I am always interested in evidences of syphilis elsewhere in the body. He did not have any chronic orchitis or perisplenitis to back up this disease in the aorta?

DR. MALLORY: I think we did not notice anything else which might be attributed to lues.

A STUDENT: Were those two nodules on the interventricular septum examined microscopically?

DR. MALLORY: Yes; they were the areas of myocarditis I mentioned. It really was a lesion of the muscular elements showing through the endocardium.

CASE 19432

THE INTERESTING RESULT OF SEVERE DEFORMITY OF THE THORAX

Medical Department

PRESENTATION OF CASE

Dr. Richard B. Capps:* This is the case of a forty-five year old clerk who entered the hospital complaining of shortness of breath of one year's duration and orthopnea of two weeks'.

A year previous to admission he had noticed the onset of attacks of what he called "nervous prostration". These were attacks of dyspnea, usually occurring at night, lasting half an hour to an hour and requiring him to sit up in bed. At times he had to go on his hands and knees in order to take a deep breath. For a while they occurred frequently; then somewhat less often. This went on until a month before admission, when he noticed moderate dyspnea on exertion which rapidly increased until he was unable to walk any distance. Two weeks before admission the attacks of dyspnea took on an asthmatic character. He said he wheezed and they were very severe. He was unable to do anything and could not lie flat. Two days before admission the asthma was so severe that he was unable to sleep.

The past history showed that from the age of a few months he had had very severe right dorsal scoliosis. Two brothers, one aunt, and one great-aunt had similar right scoliosis, though not so severe as his.

Examination on admission showed a well developed and nourished man sitting on the edge of the bed puffing and wheezing in considerable distress. He was slightly cyanotic. The spine was fixed in marked right dorsal scoliosis. There was some dullness on the right side over the prominence of the chest posteriorly. It was of doubtful significance on account of the shape of his chest. The heart was enlarged to percussion both to the right and left and of rather peculiar shape. The percussion was thought to be unreliable on account of the scoliosis. The sounds were of poor quality and the rhythm tic-tac. The blood pressure was 140/110. The neck vessels were not enlarged. The chest was full of loud bubbling râles and asthmatic wheezes and groans. The liver was probably down to the umbilicus. The edge was not felt. There were small epigastric and umbilical herniae. There was slight pitting edema of the lower legs.

The Hinton test was negative. He died about twelve hours after admission, so that no laboratory work was done while he was in the ward. In the Outpatient Department the urine showed a very slight trace of albumin, specific gravity 1.020, one or two granular casts per field, rare red cells and white cells. The blood work was said to be normal.

X-rays in the Outpatient Department showed very marked scoliosis without active disease in the chest. The heart was poorly seen. The films were taken nineteen days before admission.

At admission he was given digitalis and morphia. About twelve hours after admission he had a very severe attack of dyspnea during which he died.

*Recently senior interne on the East Medical service.

Clinical Discussion

Dr. Capps: The discharge diagnosis was right dorsal familial scoliosis, questionable mild arteriosclerotic heart disease, congestive heart failure and epigastric and umbilical herniae. It was thought the cardiac condition was due chiefly to mechanical pressure on the great vessels and heart caused by the shape of his chest.

Dr. Aubrey O. Hampton: He had so much deformity of his chest due to the scoliosis that we had our difficulties too. The heart looks a little large, but that is a guess. The scoliosis is apparently of the type that we usually see with congenital defects. We did not see enough of his right upper lobe to determine whether there was any disease there, but in the lung that we could see we could not see anything very striking. There is a shadow that I do not understand. I cannot even guess at it. I think that is the arch of the aorta, but that is also a guess.

CLINICAL DIAGNOSES (FROM HOSPITAL RECORD)

Familial scoliosis.
Congestive heart failure.
Mild arteriosclerotic heart disease.

ANATOMIC DIAGNOSES

Familial kyphoscoliosis, right dorsal.
Congestive heart failure, mechanical.
Cardiac hypertrophy and dilatation, right ventricular.

Pathologic Discussion

Dr. Tracy B. Mallory: The autopsy in this case showed as extreme a deformity as is possible in a thorax. I will read Dr. Chapman's description of it after the sternum and the thoracic viscera were removed.

"The curvature of the spine extends to the right midchest wall and the anterolateral surfaces of the vertebrae touch the ribs, but no adhesions unite them. The spine forms a huge letter S and leaves the pleural spaces markedly diminished, there being a concavity of the midthorax on the left that looks like an exaggerated Harrison's groove. From the point of extreme curvature in the right chest wall the spine turns sharply to the left and lies almost transversely until it passes the mid-

line to the left in the upper lumbar segments; it then bends back to the right to continue as the lower lumbar and sacral segments."

The aorta followed the very tortuous course of the spine. There were no definite dilatations or aneurysmal sacs. There was one spot at which the aorta was seen absolutely transversely. It was characteristic enough to have produced that spot in the plate of which Dr. Hampton spoke. Don't you think so, Dr. Chapman?

DR. EARLE M. CHAPMAN: Yes.

DR. MALLORY: The remainder of the autopsy was fairly negative except for the heart. We found nothing of significance except a marked degree of hypertrophy of the right side of the heart which would suggest a very definite obstruction in the pulmonary circuit, and the general signs of passive congestion and heart failure. So I think we have to assume that he died primarily of right heart failure, and we are almost forced to assume that the heart failure is in some respects secondary to the marked deformity of the chest, but actually the vessels of the lung show no very striking degree of arteriosclerosis.

Have you anything to add, Dr. Chapman?

DR. CHAPMAN: I think it is interesting that when this patient presented himself in the Outpatient Department he could not convince the doctors there that he was suffering from heart disease. The sounds then were apparently of good quality, the blood pressure was normal, and they turned him away with a diagnosis of possible psychoneurosis. Later he presented himself in the Emergency Ward and was treated.

DR. PAUL D. WHITE: Do you know if his diastolic pressure had been elevated before?

DR. CHAPMAN: It was normal in the Outpatient Department.

DR. WHITE: I have one or two notes of interest. Back in 1878 hypertrophy of the right ventricle in a case of high grade scoliosis was described by Traube[1]. Since then a number of articles, some rather extensive, have been written on cardiovascular complications in scoliosis. In 1923 Boas[2] reported that out of 35 cases on Virchow's service, all autopsied, Rieder found 29 cases of dilatation of the right ventricle. Bachmann[3] in 1899 reported 197 autopsied cases of scoliosis most of which showed right ventricular hypertrophy and dilatation. Out of a total of 276 cases in the literature including the 197 mentioned above, 247, or 89.5 per cent, showed organic heart disease. In 154 of these cases 87 showed right ventricular hypertrophy and dilatation and 27 left ventricular hypertrophy and dilatation. It is thus more common for the right ventricle to be under strain than the left.

The mechanism is thought to be chiefly one of increased intrapulmonary pressure or actual constriction of the aorta or other great vessels. Possibly the latter was a factor in this case, for the history of dyspnea and attacks of cardiac asthma suggested left failure rather than right.

Recently it has been quite well recognized by authorities like Coombs that fatal cardiac failure occurs commonly in older patients with severe spinal deformity. Coombs[4] reported three years ago that he had seen four striking cases all dying of congestive heart failure; they showed gallop rhythm during their terminal illness. I wonder if anyone here has noticed or had experience with such cases.

DR. CHARLES L. SHORT: I had in the Outpatient Department a patient twenty-five years old who had a severe kyphosis secondary to Pott's disease in childhood. She presented herself with cardiac symptoms which consisted in rather atypical attacks of dyspnea and palpitation. It was impossible to demonstrate by x-ray or physical examination any objective signs of cardiac disease. Furthermore there were certain things in the background which suggested a psychological mechanism for her symptoms, so she was treated with reassurance and explanation. I was notified some time later that she had died in a heart attack. After that I looked up the literature and found that this condition is well recognized. So I should like to emphasize that, with deformity of the spine, cardiac symptoms should not be taken lightly.

DR. FULLER ALBRIGHT: I saw in Vienna one striking case very much like this, with death due to right sided failure, and I remember hearing Professor Erdheim say that that was the commonest cause of death and that these patients seldom live past the age of thirty or forty.

DR. HAMPTON: There are also cases reported where the deformity of the chest and resultant cardiac failure were due to hyperparathyroidism.

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THE MENACE OF MALNUTRITION

CONSIDERABLE difficulty is entailed in evaluating accurately the effects of hard times on the physical welfare of a people. During our present critical period fears have been entertained from time to time as to the results which might be expected from a lack of food or from an unbalanced diet as related to the incidence of disease or of malnutrition. As a whole these fears have not materialized to any extent, at least in this section of the country; in fact, rather the opposite has been encountered. Epidemics have been few, the deathrate from tuberculosis has not risen, the general mortality rate has been low and the call for medical service has been far from spectacular.

It has been felt that one ill effect of the times would be their reflection on the nutritional status of the nation's children, and a year ago a local study of this vital subject was suggested. Immediately obstacles arose, chief among them being the difficulty of finding acceptable standards of nutrition on which to base a premise, and the uncertainty as to whether such malnutrition as might be found could be laid on the doorstep of the depression, and the attempt was abandoned.

Recently, however, Secretary of Labor Perkins, basing her apprehension on data gathered by the Children's Bureau of the Department of Labor, called leading health, social welfare and other experts to meet in Washington and unite on a child health-recovery program. The children, according to her announcement in the *New York Times*, "are showing signs of poor nutrition, of inadequate housing, of lack of medical care; many, too, are showing the effect of the anxiety and sense of insecurity which prevail wherever there is unemployment".

More explicitly, the data gathered by the Children's Bureau indicate that possibly one-fifth of all pre-school and school children in the country are below par in health. These data have been gathered from various types of sources; some statistical, some reports of small groups of cases, some the opinions of individuals. The report, nevertheless, is convincingly temperate in its tone, admitting that figures from one community are not necessarily applicable to another, and that because the effects of privation are demonstrable in one region it does not follow that they exist in another. The substance of it is, however, that the nutritional condition of children in many communities is showing increasingly serious effects of the long periods of unemployment and want.

In New York City figures indicate that malnutrition among school children has increased from 16 per cent in 1929 to 29 per cent in 1932 in Manhattan, and from 13 to 23 per cent in the Bronx. Detroit has shown a similar condition, and in Springfield, Ohio, the ten per cent underweight group has increased from 24 per cent in 1928 to 31 per cent in 1932. Similarly, in Philadelphia, a large group examined by the Community Health Centre has shown an increase in malnutrition from 30 per cent in the years 1928-1930 to 42 per cent in 1932. Not accurate, perhaps, these figures, and based possibly on indefinite standards, and yet they indicate a downward health trend in childhood as the shadow of the depression grows longer.

Some plan must be devised, according to Secretary Perkins, by which the undernourished children may be found and their needs relieved. Steps must be taken to provide school lunches, a diet to fit the needs of malnourished children, some special form of family relief, and as close a medical check as possible of those who appear to be malnourished.

CANCER AND OTHER CHRONIC DISEASES IN MASSACHUSETTS

A NOTABLE contribution to our understanding of the chronic disease problem is made by Drs. Bigelow and Lombard in their book, "Cancer and Other Chronic Diseases in Massachusetts." This offers the first complete study of an adequate sample of the population to be made in the field of chronic disease. While we are aware of the magnitude and the manifold ramifications of the problems of chronic illness, we are all, however, vague as to just how great the problems may be and as to what means are already available for their solution. This volume is essentially an accurate stock-taking of chronic disease in this state, together with a presentation of the facilities available for the care of the chronic patient.

As the title implies, particular attention has been directed to cancer, as is to be expected in view of the outstanding strides made by Massachusetts in this particular field.

The study includes not only data from hospitalized cases but from sample urban and rural groups as well. The adequacy of the sample is assured by the careful selection of the areas studied and the number of persons covered which is 45,487. We may fairly assume the data obtained to be accurate. On their basis we have one-half million chronic ill persons in Massachusetts. The close correlation of the incidence of chronic disease with age is vividly illustrated by these figures, the chronic disease rate being 18.5 per 1,000 for those under twenty, and 707.5 per 1,000 for those over eighty.

In these days of mounting welfare costs, the added expenditure necessitated by the care of the chronic patient is well to appreciate. In the surveyed cities, over twice as much was spent for these persons requiring welfare aid as for the healthy on the welfare rolls; and in nearly two-thirds of the families the need for help was created or aggravated by chronic illness among the members. In the population that normally would be wage-earners, one-sixth of those suffering from chronic diseases were completely disabled, and one-eighth were partially disabled.

These brief notes may serve to indicate the large amount of material contained in this study and to guide those interested in the problems of chronic disease to the vast amount of detailed information presented not only in the substance of the book itself but in the various statistical appendices.

The chapter on the economics of chronic disease is of especial interest and significance, and it closes with a suggestion of fundamental importance: "There is growing unrest among many with chronic disease, who feel that somewhere there is a service which will meet their needs, but either because of lack of resources or lack of knowledge as to where to go they are unable to obtain it. We believe that if these people realize that their own physicians can furnish at reasonable cost all that is needed, most of the unrest and dissatisfaction between the people of the medical profession will disappear[1]."

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THIS WEEK'S ISSUE

CONTAINS articles by the following named authors:

ZINSSER, HANS. A.B., A.M., Sc.D. Hon., M.D. Columbia University College of Physicians and Surgeons 1903. Associate Professor, Leland Stanford University 1910-1911. Professor, Leland Stanford University 1911-1913. Professor, Columbia University 1919-1923. Professor of Bacteriology and Immunology, Harvard University 1923-. Consulting Bacteriologist, Children's Hospital 1924-. Consultant, Peter Bent Brigham Hospital 1924-. Address: Harvard Medical School, Boston. Associated with him is

CASTANEDA, M. RUIZ. M.D. Eschula Nacional de Medicina, Mexico 1923. Assistant Mexican Department of Health 1928-. Pathologist, American Hospital, Mexico City 1929-. Instructor Harvard University 1932-. Address: Harvard Medical School, Boston. Their subject is "On the Isolation from a Case of Brill's Disease of A Typhus Strain Resembling the European Type." Page 815.

WARREN, SHIELDS. A.B., M.D. Harvard University Medical School 1923. Pathologist, New England Deaconess Hospital, New England Baptist Hospital, Huntington Memorial Hospital, Pondville State Hospital and Robert B. Brigham Hospital; Instructor, Harvard Medical School. His subject is "Studies on Tumor Metastasis. IV. Metastases of Cancer of the Stomach." Page 825. Address: 195 Pilgrim Road, Boston.

DEROW, HARRY A. M.B., M.D. Boston University School of Medicine 1927. Assistant Physician, Beth Israel Hospital. Assistant in Medicine, Harvard University Medical School. Address: 475 Commonwealth Avenue, Boston. Associated with him is

COHEN, ETHEL. B.A., M.S. Director, Social Service Department, Beth Israel Hospital, Boston. Address: Beth Israel Hospital, Boston. Their subject is "The Training of Interns in the Social Aspects of Medicine." Page 827.

JONES, EZRA A. M.D. Baltimore Medical College 1907, F.A.C.S. Orthopaedic Surgeon to

Eliot Hospital and Balch Hospital for Children, Manchester, N. H. and to St. Joseph's Hospital and Memorial Hospital, Nashua, N. H. and to Women's Memorial Hospital, Concord, N. H. His subject is "Orthopedic Treatment of Infantile Paralysis." Page 831. Address: 795 Elm Street, Manchester, N. H.

DENICOLA, PAUL. M.D. University of Vermont College of Medicine 1924. His subject is "Diagnosis and Treatment of Multiple Sclerosis." Page 834. Address: 115 Main Street, Nashua, N. H.

HOLMES, ARTHUR D. B.S., Ph.D. Johns Hopkins University, 1911. Teaching Record Laboratory Assistant, Dartmouth College, 1905-1906. Instructor in Chemistry in University of Maine 1906-1907, etc. Research Chemist, U. S. Dept. Agriculture, December 1911-1918. Now Director of Research, The E. L. Patch Company, Stoneham. Associated with him is

PIGOTT, MADELEINE G. B.S. in Chemistry, Tufts College Graduate Work in Medical Sciences. Research Assistant, Harvard Medical School and E. L. Patch Co. Address: E. L. Patch Company, Stoneham, Mass. And

BOWSER, LAWRENCE P. B.S. Senior student in Tufts College Medical School. Address: 33 Cedar Avenue, Stoneham. Their subject is "The Value of Cod Liver Oil for the Treatment of Anemia." Page 839.

WHITE, JAMES C. A.B., M.D. Harvard University Medical School 1923. F.A.C.S. Instructor and tutor in Surgery, Harvard University Medical School. Associate in Surgery, Massachusetts General Hospital, Boston. His subject is "Progress in the Surgery of the Sympathetic Nervous System in 1932." Page 843. Address: Massachusetts General Hospital, Boston.

MISCELLANY

THE MEMORIAL TO LEONARD WOOD

On October 9, in the Harvard Memorial Chapel, was unveiled a tablet to a graduate of the Harvard Medical School and a former house officer at the Boston City Hospital who forsook the art of medicine for the science of military tactics. An obscure doctor, Leonard Wood saw his strange opportunity and became a builder of armies; a far-sighted national figure, beloved in the end even by his enemies. In speaking at this ceremony Bishop Lawrence was justified in paying tribute to his "rugged force and gracious spirit, his simplicity, wisdom, love of truth and sense of honor".

AN HONOR TO DR. ZINSSER

Dr. Hans Zinsser, Professor of Bacteriology and Immunology of the Harvard Medical School, recently delivered an address before Lehigh University on the occasion of the annual Founder's Day ceremony.

In connection with the exercises an Honorary degree of Doctor of Science was conferred on Dr. Zinsser.

MORTALITY RATES

Telegraphic returns from 85 cities with a total population of thirty-seven million for the week ending September 30, indicate a mortality rate of 10.0 as against a rate of 9.4 for the corresponding week of last year. The highest rate (16.8) appears for Richmond, Va., and the lowest (6.0) for Detroit, Mich. The highest infant mortality rate (14.9) appears for New Bedford, Mass., and the lowest for Bridgeport, Conn., Kansas City, Kans., Omaha, Nebr., South Bend, Ind., Tacoma, Wash., and Waterbury, Conn., which reported no infant mortality.

The annual rate for 85 cities is 10.9 for the thirty-nine weeks of 1933, as against a rate of 11.2 for the corresponding period of the previous year.

SUMMARY OF DEATHS AND DEATHRATES (ANNUAL BASIS) FROM AUTOMOBILE ACCIDENTS PER 100,000 ESTIMATED POPULATION FOR 86 CITIES FOR CORRESPONDING PERIODS OF 1933 AND 1932

	Week ending Sept. 30, 1933	Week ending Oct. 1, 1932	First 39 weeks 1933	First 39 weeks 1932
Total deaths	179	166	5,684	5,658
Deathrate	24.9	23.6	20.3	20.6
Deaths due to accidents in city	138	135	4,467	4,568
Deathrate	19.2	19.2	16.0	16.6

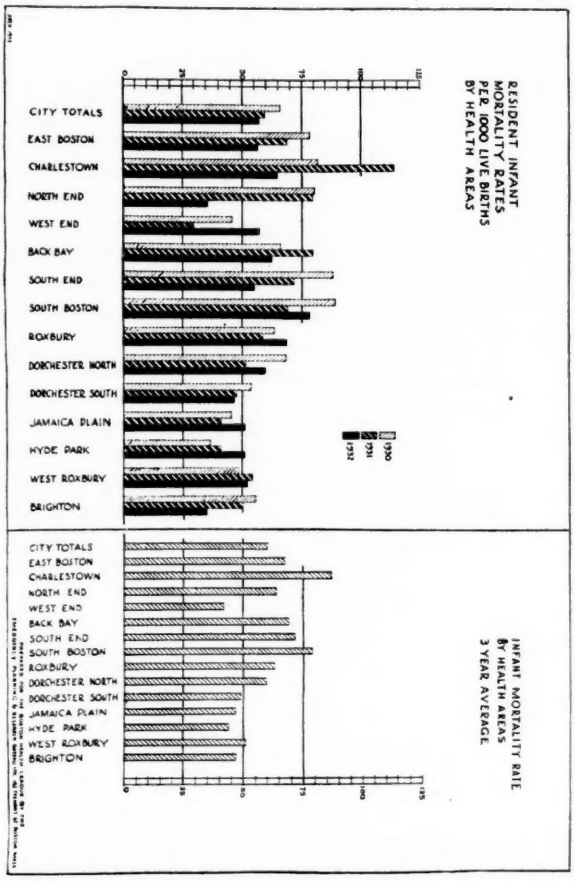
A STUDY OF INFANT MORTALITY BY HEALTH AREAS AND CENSUS TRACTS FOR 1932

PREPARED BY THE BOSTON HEALTH LEAGUE, INC.

The Boston Health League has completed its tabulation of resident births and infant deaths for 1932 by Health Areas and Census Tracts, which is the third year for this series of figures. It is interesting to note that the infant mortality rate has declined steadily during these years, being 66.6 for 1930, 59.4 for 1931, and 57.5 for 1932, the lowest rate on record. The number of births in 1930 was 13,894 and declined to 12,975 in 1931, then increased slightly in 1932 being 13,092. Stillbirths remained relatively the same, being 446, 403, and 427 for the three years.

The graph accompanying this table shows the infant mortality rate for each of these three years by Health Areas and the three-year average for each area. It is interesting that the average for Charlestown is much above the city average, while the West End is very much below, in fact the highest and lowest rates in the city. Since these areas are comparable in economic status, the median monthly rental in Charlestown was $22.00

and in the West End $28.00 at the time of the 1930 census, while the density of population in Charlestown was 255.8 persons per inhabited land acre and 333.7 persons in the West End, the Health League was interested in determining the factors causing these rates, if possible. A joint infant mortality study is therefore being conducted for these two areas. The Harvard School of Public Health is making a medical investigation for



each resident infant death occurring in these districts during 1933, while the Bureau of Research of the Boston Council of Social Agencies is doing the social investigation for each.

While the number of infant deaths in any one of these areas is relatively small in a given year, it is felt that the three-year average is rather a good indication of conditions, although we realize that the five-year average will be of greater value than the three.

Boston Health League

Resident Births, Still Births, Infant Deaths and Infant Mortality Rates for 1932 Distributed by Health Areas and Census Tracts

Census Tract	Births	Deaths Under One Year	Infant Mortality Rate	Still Births	First Day	First Month	After First Month
City Totals	13,092	753	57.5	427	219	208	326
East Boston							
A-1	87	3	34.5	5	2	—	1
A-2	73	5	68.5	3	1	1	3
A-3	168	9	53.6	8	3	3	3
A-4	111	6	54.0	1	1	4	1
A-5	163	7	42.9	5	1	1	5
A-6	117	8	68.4	5	1	3	4
B-1	93	3	32.3	1	—	1	2
B-2	92	6	65.2	3	1	1	4
B-3	66	5	75.8	—	3	1	1
B-4	74	3	40.5	6	—	2	1
B-5	183	15	82.0	7	4	5	6
	1,227	70	57.0	44	17	22	31
Charlestown							
C-1	32	3	93.7	—	1	—	2
C-2	82	8	97.6	3	1	1	6
C-3	84	6	71.4	5	1	1	4
D-1	25	—	—	2	—	—	—
D-2	22	—	—	1	—	—	—
D-3	70	3	42.9	2	2	—	1
D-4	67	6	89.5	2	2	3	1
E-1	89	6	67.4	1	1	3	2
E-2	44	2	45.4	1	—	—	2
	515	34	66.0	17	8	8	18
North End							
F-1	101	5	49.5	2	1	1	3
F-2	112	4	35.7	4	2	1	1
F-3	20	1	50.0	2	—	—	1
F-4	128	3	23.4	3	—	2	1
F-5	99	4	40.4	1	1	2	1
F-6	8	—	—	—	—	—	—
	468	17	36.3	12	4	6	7
West End							
H-1	128	10	78.1	4	—	3	7
H-2	65	3	46.1	3	1	1	1
H-3	31	1	32.3	—	1	—	—
H-4	98	8	81.6	3	2	3	3
K-1	60	1	16.7	2	—	—	1
K-2	32	1	31.2	—	—	—	1
	414	24	58.0	12	4	7	13
Back Bay							
K-3	9	3	333.3	—	—	1	2
K-4	96	4	41.7	3	3	1	—
K-5	15	—	—	—	—	—	—
J-3	25	3	120.0	—	2	—	1
J-4	54	5	92.6	1	2	3	—
J-5	70	5	71.4	2	1	4	—
S-1	79	2	25.3	3	—	—	2
	348	22	63.2	9	8	9	5
South End							
G-1	35	1	28.6	1	—	—	1
G-2	50	1	20.0	2	—	1	—

G-3	7	—	—	—	—	—	—
G-4	8	2	250.0	—	—	—	2
I-1	49	2	40.8	1	2	—	—
I-2	94	4	42.5	—	1	—	3
I-3	76	2	26.3	—	—	1	1
I-4	60	6	100.0	3	3	—	3
J-1	46	2	43.5	1	—	2	—
J-2	24	1	41.7	1	—	—	1
L-1	62	3	48.4	1	3	—	—
L-2	63	6	95.2	4	2	3	1
L-3	67	10	149.2	5	1	4	5
L-4	49	—	—	1	—	—	—
L-5	42	4	95.2	1	—	1	3
L-6	49	1	20.4	2	—	—	1
Q-1	50	1	20.0	1	1	—	—
	831	46	55.3	24	13	12	21

South Boston

M-1	52	8	153.8	3	3	2	3
M-2	76	5	65.8	2	—	2	3
M-3	47	12	255.3	2	2	6	4
M-4	36	1	27.8	1	—	—	1
N-1	137	19	138.7	7	1	6	12
N-2	83	—	—	2	—	—	—
N-3	80	6	75.0	3	3	—	3
N-4	83	1	12.0	7	—	—	1
O-1	159	13	81.7	5	5	3	5
O-2	73	6	82.2	1	2	1	3
O-3	81	5	61.7	5	1	—	4
O-4	36	1	27.8	2	—	—	1
P-1	108	6	55.5	4	1	2	3
	1,051	83	79.0	44	18	22	43

Roxbury

Q-2	106	5	47.2	4	1	1	3
Q-3	77	10	129.9	—	3	2	5
Q-4	95	5	52.6	1	—	1	4
R-1	131	12	91.6	6	1	2	9
R-2	84	5	59.5	3	1	—	4
R-3	74	5	67.6	6	1	1	3
S-2	50	7	140.0	—	2	2	3
S-3	110	6	54.5	7	3	2	1
S-4	79	7	88.6	1	1	2	4
S-5	85	4	47.1	3	1	—	3
S-6	65	3	46.1	—	1	1	1
U-1	136	6	44.1	6	4	2	—
U-2	117	8	68.4	3	2	2	4
U-3	87	8	91.9	2	2	1	5
U-4	124	12	96.8	3	7	—	5
U-5	143	9	62.9	5	4	1	4
U-6	197	7	35.5	2	4	1	2
V-1	84	5	59.5	1	—	1	4
V-2	112	11	98.2	3	3	4	4
	1,956	135	69.0	56	41	26	68

Dorchester North

P-2	99	4	40.4	5	—	2	2
P-3	70	6	85.7	3	1	2	3
P-4	91	9	98.9	6	5	1	3
P-5	78	7	89.7	3	2	1	4
P-6	60	2	33.3	3	1	1	—
Q-5	144	16	111.1	8	5	3	8
T-1	101	5	49.5	6	3	1	1
T-2	125	8	64.0	2	3	1	4
T-3	143	8	55.9	11	2	1	5
T-4	181	8	44.2	3	2	2	4
T-5	197	17	86.3	7	5	6	6
T-6	87	7	80.5	6	4	—	3
T-7	171	5	29.2	4	2	—	3
T-8	136	3	22.1	2	1	2	—
T-9	99	5	50.5	4	1	3	1
T-10	60	3	50.0	—	—	1	2
X-1	194	10	51.5	13	3	5	2
	2,036	123	60.4	86	40	32	51

Dorchester South

X-2	192	9	46.9	9	5	1	3
X-3	187	12	64.2	10	5	6	1
X-4	220	11	50.0	9	5	3	3
X-5	311	12	38.6	4	4	4	4
X-6	322	14	43.5	12	2	3	9
	1,232	58	47.1	44	21	17	20

Jamaica Plain

V-3	98	5	51.0	5	1	2	2
V-4	88	5	56.8	1	1	1	3
V-5	129	9	69.8	2	3	3	3
V-6	145	8	55.2	5	3	2	3
W-1	192	10	52.1	4	7	—	3
W-2	135	4	29.6	1	3	1	—
	787	41	52.1	18	18	9	14

Hyde Park

Z-1	307	16	52.1	7	6	6	4
Z-2	135	7	51.8	2	1	3	3
	442	23	52.0	9	7	9	7

West Roxbury

W-3	180	10	55.5	3	2	2	6
W-4	191	13	68.1	9	6	6	1
W-5	143	7	48.9	5	1	3	3
W-6	303	13	42.9	3	3	5	5
	817	43	52.6	20	12	16	15

Brighton

Y-1	119	1	8.4	2	—	—	1
Y-2	113	6	53.1	5	1	4	1
Y-3	245	11	44.9	4	2	4	5
Y-4	149	3	20.1	5	1	2	—
Y-5	341	13	38.1	16	4	3	6
	967	34	35.1	32	8	13	13
B-6	1	—	—	—	—	—	—

THE NOMINATION OF DR. BUDRESKI

Governor Ely on October 4, 1933, nominated Dr. Alphonse F. Budreski of Brockton to be medical examiner to the first Plymouth District to succeed Dr. Walter W. Fullerton of Brockton whose term had expired.

HOME NURSING SERVICE FOR LOBAR PNEUMONIA CASES

Clinical experience indicates that the best chances of recovery obtain among pneumonia patients who receive prompt medical and nursing care. The Metropolitan Life Insurance Company is, in consequence, desirous of stimulating a coördinated effort in Boston in order that patients with lobar pneumonia may receive adequate medical and nursing care as early as possible. It is desirous of coöperating with the family physicians to this end, and it is prepared to give nursing care to its industrial policyholders through the Community Health Association. Nursing service through this Association is also available at cost for any family in Boston needing this care. If the family is unable to pay, free service is given. Nursing care is given only under the order of an attending physician.

The demonstration will cover a period of one year (November 1, 1933-November 1, 1934).

Physicians of the Norfolk and Suffolk County Medical Societies are asked to coöperate by:

1. Reporting all pneumonia cases to the City Health Department promptly.
2. Calling the Community Health Association which is prepared to offer nursing service on a visit basis to those patients not needing or unable to employ a full-time nurse.

Lobar pneumonia, the most serious of all the acute infectious diseases, has taken an annual toll of lives with little apparent variation from year to year. In Boston during 1932, 1013 cases were reported with 437 deaths. The disease is at present the seventh leading cause of death in the city. A survey by the Boston Health League indicates that the number of reported cases and the deaths is in direct proportion to the density of the population in the city.

Investigations of lobar pneumonia among policyholders in the Metropolitan Life Insurance Company in various parts of the United States indicate that nurses reach only about ten per cent of the fatal cases. Eliminating those admitted to hospitals and those who die suddenly there remains about fifty per cent of the fatal cases who could and should have had nursing service and did not.

The Boston Health League is assisting in the coördination of this demonstration and the following are serving as the Medical Advisory Committee:

Frederick T. Lord, M.D., Chairman
Frank S. Cruickshank, M.D.
Dwight O'Hara, M.D.
Wilson G. Smillie, M.D., Dr.P.H.

Physicians' attention is called to the fact that *this demonstration is confined to municipal Boston.* For any further information please communicate with the Boston Health League, 43 Tremont Street; Lafayette 6566.

AN HONOR TO DR. MILTON J. ROSENAU

At the meeting of the American Public Health Association in Indianapolis, October 9, 1933, the Sedgwick Memorial Medal for distinguished service in Public Health was awarded to Dr. Milton J. Rosenau of Harvard University.

The Presentation Speech by Professor Edwin O. Jordan was as follows:

It is my privilege, as Chairman of the Committee on the Sedgwick Memorial Medal, to announce the award of this medal for distinguished service in public health to Milton Joseph Rosenau of the Harvard Medical School. The award is made on the basis of Dr. Rosenau's long and fruitful career in the field of public health, for his remarkably successful ten-year administration of the Hygienic Laboratory of the U. S. Public Health Service where he carried out pioneer investigations on anaphylaxis, for his practical work on disinfectants and on the pasteurization of milk, for his numerous original contributions to bacteriology and epidemiology, for his wise and inspiring participation in coöperative research upon influenza, pneumonia and poliomyelitis, and especially for the service he has rendered public health workers throughout the world by his classic book on Preventive Medicine. This award is peculiarly appropriate since Dr. Rosenau and Professor Sedgwick were intimately associated in the organization and direction of the pioneer Harvard-Technology School of Public Health.

CORRESPONDENCE

ARTICLES ACCEPTED BY THE AMERICAN MEDICAL ASSOCIATION COUNCIL ON PHARMACY AND CHEMISTRY

535 North Dearborn Street, Chicago, Ill.,
October 4, 1933.

Managing Editor,
The *New England Journal of Medicine,*

In addition to the articles enumerated in our letter of August 31 the following have been accepted:

Abbott Laboratories
Abbott's Haliver Oil, Plain
Chloriodized Rapeseed Oil
Ampules Campiodol Emulsion, 20 cc.

Gilliland Laboratories
Staphylococcus Vaccine (Albus and Aureus)
5 cc. vial

Adolphe Hurst & Co.
Metaphyllin
Ampules Solution Metaphyllin, 0.24 Gm., 10 cc.
Ampules Solution Metaphyllin, 0.48 Gm., 2 cc.
Suppositories Metaphyllin, 0.36 Gm.
Tablets Metaphyllin, 0.1 Gm.

Lederle Laboratories, Inc.
Fish Glue Allergenic Extract (Lederle)

Mead Johnson & Co.
Mead's Halibut Liver Oil
Mead's Halibut Liver Oil with Viosterol 250 D.

Parke, Davis & Co.
Parke-Davis Haliver Oil, Plain

Petrolagar Laboratories
Petrolagar (with Cascara, Non-Bitter)

Pharmedic Corporation
Aminophylline—Pharmedic
Ampules Solution Aminophylline — Pharmedic, 0.24 Gm., 10 cc.
Ampules Solution Aminophylline — Pharmedic, 0.48 Gm., 2 cc.
Suppositories Aminophylline — Pharmedic, 0.36 Gm.
Tablets Aminophylline—Pharmedic, 0.1 Gm.

Radium Chemical Co.
Radium Chloride—Radium Belge

G. D. Searle & Co.
Aminophylline—Searle
Ampules Solution Aminophylline — Searle, 0.24 Gm., 10 cc.
Ampules Solution Aminophylline — Searle, 0.48 Gm., 2 cc.
Tablets Aminophylline — Searle, 0.1 Gm. (1½ grains)

Smith, Kline & French Laboratories
Benzedrine
Benzedrine Inhaler
Benzedrine Solution

E. R. Squibb & Sons
Autolyzed Liver Concentrate—Squibb
False Ragweeds Combined Pollen Allergen Solution — Squibb (False Ragweed and Slender Ragweed in equal parts); Orachs (Shadscale) Pollen Allergen Solution—Squibb (Shadscale, Redscale and Wingscale in equal parts); Oregon Ash Pollen Allergen Solution—Squibb; Ragweed Combined Pollen Allergen Solution — Squibb (Giant Ragweed and Dwarf Ragweed in equal parts); Rye Grasses Combined Pollen Allergen Solution — Squibb (Perennial Rye Grass and Italian Rye Grass in equal parts); Sagebrush Combined Pollen Allergen Solution—Squibb (Sagebrush and Pasture Sage in equal parts); Wormwoods Combined Pollen Allergen Solution—Squibb (Biennial Wormwood, Dragon Sagewort, Dark-leaved Mugwort, and Mugwort in equal parts).
Squibb Stabilized Refined Halibut-Liver Oil
Squibb Stabilized Refined Halibut-Liver Oil with Viosterol 250 D

The following product has been included in the List of Articles and Brands Accepted by the Council But Not Described in N.N.R. (New and Non-official Remedies, 1933, p. 437):

Lederle Laboratories, Inc.
Fish Glue Allergenic Extract Glycerinated (Lederle)

Yours sincerely,
PAUL NICHOLAS LEECH, *Secretary*,
Council on Pharmacy and Chemistry.

A STOLEN MICROSCOPE

September 29, 1933.

Dr. Walter L. Burrage, Secretary,
Massachusetts Medical Society,
182 Walnut Street, Brookline, Mass.

Dear Doctor:

I am 53 years old, the sole support of a family of seven members, a practitioner since 1905, a Fellow of the Massachusetts Medical Society. I have practiced in Boston since 1921. I was badly hit by the depression. In the night of September 15-16, a burglar entered my office and stole my microscope, on which I depended mostly for the routine of my practice.

It was a new Leitz microscope, bought in 1924, No. 219423, with 3 objectives, oil (fluorid), high and low eye pieces and complete appendages with a rectangular stage which was somewhat discolored, being swabbed with xylol.

I have applied to the police, instrument dealers and medical schools for help in tracing it. I have not heard anything encouraging about finding it.

I had paid over $200.00 for it. Now this loss is a big blow to my practice, as I am positively unable even to spend money for its tracing.

Is it not possible for you to help me to trace it through the channel of the Massachusetts Medical Society?

Respectfully yours,
N. M. BAGHDOYAN.

331 Massachusetts Avenue,
Boston, Mass.

RECENT DEATHS

CURRIER — WILLIAM HALE CURRIER, M.D., of Worcester, Mass., died in that city September 28, 1933. Dr. Currier was born in Vermont in 1859, the son of Samuel H. and Abbie (Kimball) Currier. He graduated in medicine from the Dartmouth Medical School in 1881 and practiced in Hartford, Conn., Vermont, and Pittsfield before taking residence in Worcester where he practiced for the past five years. He joined the Massachusetts

Medical Society in 1890 and was also a member of the American Medical Association. He was affiliated with the Masonic order. He is survived by his widow Mrs. Edna H. (Legg) Currier.

FISH—RALPH CHARLES FISH, M.D., of Worcester, Mass., died in that city September 28, 1933. Dr. Fish was born in Preston, England, the son of Timothy and Elizabeth (Isherwood) Fish and came to this country in his early years and lived for a time in St. Louis and later in New Bedford and Fairhaven. He graduated in medicine from the Baltimore Medical College in 1894 and after serving two years in the West London Hospital began practice in Worcester. He joined the Massachusetts Medical Society in 1902. His membership terminated in 1924. He was a Mason and also affiliated with the First Unitarian Church. He is survived by his widow Mrs. Bertha E. (Record) Fish, a daughter Mrs. Richard L. Kimball of Wilkinsburg, Pa., and a son Preston R. Fish of Worcester.

BARROWS — ELMA ASHLEY BARROWS, M.D., of Plymouth, Massachusetts, died in that town October 18, 1933. He was born in Vermont in 1870, and graduated in medicine from the Dartmouth Medical School in 1900. Previous to settling in Plymouth, he practiced for several years in New Hampshire and Vermont.

He was a member of the Aleppo Shrine of Boston, and the Plymouth Lodge of Elks. He is survived by his widow, a daughter, Miss Vera Barrows, and son, Gordon Barrows.

BUTLER — SAMUEL BUTLER, M.D., of West Medway, Massachusetts, died in that town, October 16, 1933, where he had practiced for thirty years. He was born in 1879, and graduated in medicine from the Baltimore University Medical School in 1904. He served overseas in the World War, and later was state surgeon of the Massachusetts Division of Veterans of Foreign Wars. He joined the Massachusetts Medical Society in 1917.

NOTICES

RADIO HEALTH MESSAGES

OCTOBER-NOVEMBER-DECEMBER, 1933

HEALTH MESSAGES

Sponsorship: Public Education Committee of the Massachusetts Medical Society and Massachusetts Department of Public Health.

Courtesy WBZ. Fridays, 4:30 P.M.

Oct.
27 Rheumatism—Then and Now

Nov.
3 Danger Signals in the Rheumatic Infections of Childhood
10 Communicable Diseases in the Schools
17 Illness and Character
24 Heart Disease in Middle Age

Dec.
1 The Cause and Treatment of Crossed Eyes
8 Infantile Paralysis
15 The Preschool Child
22 Cancer of the Intestinal Tract
29 Preventive Medicine from Your Family Physician

STATE HOUSE BROADCAST

Sponsored by the Massachusetts Department of Public Health.

Courtesy WEEI. Fridays, 1:15 P.M.

Health Subjects of Public Interest discussed by the Division Directors of the Department.

RADIO HEALTH FORUM

Queries from the public are answered under the sponsorship of the Department of Public Health.

Courtesy WEEI. Fridays, 5:00 P.M.

Questions on Health and Prevention of Disease may be sent to Radio Health Forum, State Department of Public Health, State House, Boston.

CLINIC AT THE PETER BENT BRIGHAM HOSPITAL

On Thursday, November 2, in the amphitheatre of the Peter Bent Brigham Hospital at 3:30 P.M. there will be a clinic conducted by Dr. Henry A. Christian, Hersey Professor of the Theory and Practice of Physic, in the Harvard Medical School, to which physicians are cordially invited.

A CORRECTION

House of the Good Samaritan
Boston

October 19, 1933.

Editor, *New England Journal of Medicine*,

May I call your attention to a misprint in connection with the visiting activities of Dr. Howard B. Sprague as outlined in your October 5 *Journal*. He is listed as "Visiting Physician and Research Director of the House of the Good Samaritan." Dr. T. Duckett Jones is the Research Director and not Dr. Howard B. Sprague, who is a visiting physician to this hospital and has been acting as adviser of the Research Department.

Sincerely yours,

L. M. COLEMAN, *Superintendent.*

THE RESIGNATIONS OF DR. CHARLES H. LAWRENCE

Dr. Charles H. Lawrence has resigned from the staff of the Evans Memorial Hospital for clinical research. He has also resigned his position as assistant professor of Endocrinology at the Boston University Medical School.

DR. MERRITT TO SPEAK AT THE STATE HOSPITAL FOR MENTAL DISEASES, HOWARD, R. I.

Dr. H. Houston Merritt of the Department of Neuropathology of the Harvard Medical School will speak at the State Hospital for Mental Diseases, Howard, R. I., on "Diffused Sclerosis" on Monday night, October 30, 1933, at 8:15 P.M.

JACOB KASANIN, M.D., *Clinical Director.*

ANNOUNCEMENT

BRYANT D. WETHERELL, M.D., who has been associated with Dr. Arthur L. Chute for the past 14 years, announces the opening of his office at 520 Commonwealth Avenue, Boston.

REMOVALS

EDWARD F. MURPHY, M.D., announces the removal of his office to 394 Riverway, corner of Huntington Avenue at South Huntington Avenue, Roxbury.

TRACY JACKSON PUTNAM, M.D., announces the removal of his office to the Massachusetts General Hospital (Bulfinch Building), Boston.

JOHN J. DUMPHY, M.D., announces the removal of his office to Suite 510 Slater Building, Worcester, Mass.

REPORTS AND NOTICES OF MEETINGS

ETHER DAY AT THE MASSACHUSETTS GENERAL HOSPITAL

Ether Day was celebrated at the Massachusetts General Hospital on October 16, 1933, at 4:00 P.M. in the Moseley Memorial Building.

Mrs. Nathaniel Thayer, a member of the Board of Trustees, presided.

Dr. Leroy M. S. Miner gave an address.

Dr. George H. Wright was the Chairman in charge of the program.

After Dr. Miner's address the moving picture film "The Advent of Anesthesia" was shown, following which tea was served.

About six hundred people, including physicians, dentists, and friends of the Hospital, attended.

NEW ENGLAND OBSTETRICAL & GYNECOLOGICAL SOCIETY

The fifth annual meeting of the New England Obstetrical & Gynecological Society is to take place in Boston, on Wednesday, November 22, 1933.

Clinics will be held in the morning at the Women's Free Hospital in Brookline, and the Boston City Hospital, and in the afternoon at the Boston Lying-In Hospital and the Carney Hospital. The speaker of the evening will be announced at a later date.

FRED. J. LYNCH, M.D., *Secretary.*

THE NORFOLK DISTRICT MEDICAL SOCIETY

A stated meeting of the Society will be held in the Hotel Kenmore, Tuesday evening, October 31, 1933, at 8:15 P.M. Telephone Ken. 2770.

Order of Business:

1. Reading of records of previous meeting.
2. Reports from Committees.
3. Incidental business.

Action on proposed new By-Laws and such other business as may come before the meeting.

Communication:

"Paranasal Sinus Disease and the General Practitioner." Diagnosis and Treatment. Dr. H. J. Inglis.

Collation.

FRANK S. CRUICKSHANK, M.D., *Secretary.*
1695 Beacon Street.

NEW ENGLAND HEART ASSOCIATION

The New England Heart Association is planning to combine during the coming winter the monthly meetings, which have been held for the past three years on Wednesday afternoons at the Boston Medical Library, with the Cardiac Research Meetings which have been held in various hospitals at irregular intervals throughout the winter months. The first one will be held in the

ETHER DOME AT THE MASSACHUSETTS GENERAL HOSPITAL

Monday, October 30, at 8:15 P.M.

Other meetings will be held in different hospitals on the last Monday of each month from November through May, except the December meeting which will be held on the eighteenth. All members of the New England Heart Association and physicians interested are invited to attend. Members of the New England Heart Association will be notified by mail of the meeting places prior to each meeting.

PAUL W. EMERSON, M.D., *Secretary.*

BOSTON MEDICAL HISTORY CLUB

The Boston Medical History Club, organized by a few physicians interested in the subject in 1921, meets six times a year on the third Monday of the month, beginning in November. The Club is open to all physicians, medical students or laymen and the officers hope that anyone who desires to come to any of the meetings will do so, whether a member of the organization or not. Membership

may be obtained, however, by applying to the secretary, Mr. James F. Ballard, at the Boston Medical Library. There are small annual dues, used to defray, in part, the expense of obtaining out-of-town speakers.

PROGRAM

November 19, 1933: Medical-Historical Pageant under direction of Dr. B. Spector.

December 17, 1933: Prof. Norman E. Himes, of Colgate University, "Medical History of Contraception."

January 15, 1934: Prof. Henry E. Sigerist, Director of the Institute of Medical History of Johns Hopkins University.

FAULKNER HOSPITAL CLINICAL MEETINGS

On Thursday afternoon, October 5, at five o'clock, the first of the clinical meetings for the present winter months was held at The Faulkner Hospital. Four interesting cases were taken up for discussion by members of the clinical staff, x-ray findings were demonstrated by the roentgenologist and the autopsy findings reported. A discussion was then held to emphasize the lessons that might be learned from these cases.

The first case was one of coronary occlusion with extensive destruction of the wall of the left ventricle followed by a fall in blood pressure and a marked diminution in the excretion of urine by a kidney which had previously been slightly damaged by chronic vascular disease so that the patient died of uremia.

The second case was one of lymphogranulomatosis and quite similar to Hodgkin's disease. The neoplasm was distributed chiefly in the retroperitoneal and mediastinal tissues with extensive involvement of the liver and spleen. The clinical picture was most interesting because two years before the death of the patient a gland had been removed from the neck for biopsy and the diagnosis of probable tuberculosis was made. About eight months before death, because of pain in the back, the question of tuberculosis of the spine was raised and about five months before the patient died an operation on the spine, because of the suspicion of osteomyelitis of the vertebra, was performed. The instructive points in the case were the importance of continued fever in a case of lymphoid malignancy and the confusion between this condition and tuberculosis at biopsy.

The third case was one of carcinoma of the bladder which finally shut off the ureters causing uremia combined with chronic diverticulitis, with a history of a past and recent ischiorectal abscess. In this case there was evidence of chronic inflammation and scar tissue extending along the ureters and surrounding the kidneys. The point which even the autopsy could not settle was whether the chronic inflammation along the outside of the urinary tract was the result of infection through the carcinoma of the bladder or from the chronically inflamed diverticula.

The fourth case was one of very extensive formation of stones in the pelves of both kidneys in a young individual, twenty-six years of age, with enough destruction of the kidneys to cause uremia. The lesson learned from this case was that attention should be paid to the microscopic study of the urine in a patient who is running a slight elevation of temperature and feeling below par and that the patient should not be discharged until the urinary findings have cleared up and a definite diagnosis made.

The next meeting at The Faulkner Hospital will be held at 5:00 P.M. on Thursday, November 2. At that meeting there will be a clinical pathological discussion of the interesting cases which have been autopsied during the month and Dr. J. Stewart Rooney will give a short outline of what the practicing physicians and surgeons may secure from the laboratory in the way of help in their practice without actually having the patients stay in the hospital for observation. All physicians who are interested are cordially invited.

SOCIETY MEETINGS, CONGRESSES AND CONFERENCES

October 26—The Physicians Alliance will meet at 8:15 P.M. at the Whittier Street Health Unit, Roxbury. Dr. Elliott C. Cutler will be the guest speaker.

October 27—Massachusetts Psychiatric Society will meet at 8:15 P.M. at the Hotel Somerset, Boston.

October 28—The American Board of Dermatology and Syphilology will hold written examinations for candidates in the following cities: New York City; St. Louis, Mo.; Boston, Mass.; Cleveland, Ohio; San Francisco, Calif.; Philadelphia, Pa.; Chicago, Ill. Information may be obtained from the Secretary, Dr. C. Guy Lane, 416 Marlborough Street, Boston, Mass.

October 30—New England Heart Association. See page 864.

November 2—Clinic at the Peter Bent Brigham Hospital. See page 863.

November 2—Clinical Meeting at the Faulkner Hospital, 5 P.M. See notice above.

November 10—William Harvey Society will meet at the Beth Israel Hospital at 8 P.M.

November 19, December 17, January 15—Boston Medical History Club. See page 864.

November 22—New England Obstetrical and Gynecological Society. See page 864.

December 15 and 16—The American Board of Dermatology and Syphilology will hold oral examinations in New York City. Information may be obtained from the Secretary, Dr. C. Guy Lane, 416 Marlborough Street, Boston, Mass.

April 16-20, 1934—The American College of Physicians will hold its Eighteenth Annual Clinical Session in Chicago at the Palmer House. For information write Mr. E. R. Loveland, Executive Secretary, 133-135 South 36th Street, Philadelphia, Pa.

July 24-31, 1934—The IVth International Congress of Radiology will be held in Zurich under the presidency of Professor H. R. Schniz. General Secretary Dr. H. E. Walther, Gloriastrasse 14, Zurich.

September 4, 5, 6, 1934—International Union Against Tuberculosis will be held in Warsaw. For particulars address The National Tuberculosis Association, 450 Seventh Avenue, New York, N. Y.

DISTRICT MEDICAL SOCIETIES

ESSEX NORTH DISTRICT MEDICAL SOCIETY

November 2—Censors' Meeting will be held at Hotel Bartlett, Main Street, Haverhill, at 4 P.M.

E. S. BAGNALL, M.D., Secretary-Treasurer.
281 Main Street, Groveland, Mass.

ESSEX SOUTH DISTRICT MEDICAL SOCIETY

Thursday, November 2—Censors' Meeting at Salem Hospital, 3:30 P.M.

Wednesday, November 8—Salem Hospital. Clinic 5 P.M. Dinner 7 P.M. Speaker: Dr. John Lovett Morse, Boston. Subject: to be announced.

Wednesday, December 6—Beverly Hospital. Clinic 5 P.M. Dinner 7 P.M. Speaker: Dr. Hans Zinsser, Harvard Medical School. Subject: Bubonic Plague.

Wednesday, January 3—Danvers State Hospital. Clinic 5 P.M. Dinner 7 P.M. Speaker: Dr. Clarence A. Bonner, Superintendent of Danvers State Hospital. Subject: Recent Experiences in Foreign Clinics.

February 7—Council Meeting, Boston.

Wednesday, March 7—Lynn Hospital. Clinic 5 P.M. Dinner 7 P.M. Speaker: Dr. Frank H. Lahey, Boston. Subject: to be announced. Film Electrocardiogram.

Wednesday, April 4—Essex Sanatorium, Middleton. Clinic 5 P.M. Dinner 7 P.M. Speaker: to be announced. Subject: to be announced.

Thursday, May 3—Censors' Meeting, at Salem Hospital, 3:30 P.M.

Tuesday, May 8—Annual Meeting. Salem Country Club, Forrest Street, Peabody. Dinner at 7. Speaker: to be announced. Subject: to be announced.

RALPH E. STONE, M.D., Secretary.

221 Cabot Street, Beverly, Mass.

FRANKLIN DISTRICT MEDICAL SOCIETY

Meetings will be held on the second Tuesday of November, January, March and May at the Weldon Hotel, Greenfield, at 11 A.M.

CHARLES MOLINE, M.D., Secretary.

Sunderland, Mass.

MIDDLESEX EAST DISTRICT MEDICAL SOCIETY

Schedule 1933-1934

Meetings will take place on November 8 at the Bear Hill Club, Stoneham; January 10 at Melrose; March (2nd Wednesday) at Wakefield, and May (2nd Wednesday) at Winchester.

ALLAN R. CUNNINGHAM, M.D., Secretary.

76 Church Street, Winchester, Mass.

MIDDLESEX NORTH DISTRICT MEDICAL SOCIETY

Meetings will be held on January 31, and April 25 (Annual Meeting).

T. A. STAMAS, M.D., Secretary.

226 Central Street, Lowell, Mass.

NORFOLK DISTRICT MEDICAL SOCIETY

October 31—Stated Meeting, Hotel Kenmore, 8 P.M. See page 864.

November 28—Roxbury Masonic Temple, 8 P.M. Pneumonia Symposium, in charge of Dr. Frederick T. Lord.

January 28—Tentatively Roxbury Masonic Temple, 8 P.M. Continuance of Symposium on Diseases of the Blood. Dr. Dameshek and Dr. Straus.

February 27—Place, time, subject, and speaker to be announced.

March 27—Place and time to be announced. Dr. Edward A. Edwards and Dr. Henry H. Faxon. Symposium on the Treatment of Varicose Veins.

May—Annual Meeting. Place, time and program to be announced.

FRANK S. CRUICKSHANK, M.D., Secretary.

1695 Beacon Street, Brookline, Mass.

NORFOLK SOUTH DISTRICT MEDICAL SOCIETY

November 2—12 noon at Norfolk County Hospital. Stated Meeting. Speaker: Dr. Timothy Leary. Subject: Comparative Experimental Arteriosclerosis with Human Coronary Sclerosis.

December 7—12 noon at Quincy City Hospital. Program by the hospital staff.

January 4—12 noon at Norfolk County Hospital. Speaker to be announced.

February 1—12 noon at Norfolk County Hospital. Stated Meeting. Speaker: Dr. Sara Jordan. Subject: Stomach Ulcers.

March 1—12 noon at Quincy City Hospital. Program by the hospital staff.

April 5—12 noon at Norfolk County Hospital. Speaker: Dr. Elliott P. Joslin. Subject: Diabetes.

May 3—12 noon at Norfolk County Hospital. Annual Meeting. Election of Officers.

N. R. PILLSBURY, M.D., Secretary.

Norfolk County Hospital, South Braintree, Mass.

SUFFOLK DISTRICT MEDICAL SOCIETY

November 2—Censors' Meeting will be held at the Boston Medical Library, 8 Fenway, at 4 o'clock.

November 29—Clinical Program at the Beth Israel Hospital.

January 31—General Meeting in association with the Boston Medical Library and the Trudeau Society. Speaker: Dr. Lawrason Brown of Saranac Lake.

March 28—Clinical Meeting at the Massachusetts Memorial Hospitals.

April 25—Annual Meeting at the Boston Medical Library. Election of Officers. Scientific Program and titles and speakers to be announced.

The Medical Profession is cordially invited to attend all of these meetings.

JAMES H. MEANS, M.D., Vice-President.
GEORGE P. REYNOLDS, M.D., Secretary.
311 Beacon Street, Boston, Mass.
ALEXANDER S. BEGG, M.D.,
Boston Medical Library.

WORCESTER DISTRICT MEDICAL SOCIETY

All meetings to be held on Wednesdays as follows:

November 8—Dinner and scientific program at the Grafton State Hospital, North Grafton, Mass.

December 13—Dinner and scientific program at St. Vincent Hospital, Worcester, Mass.

January 10—Dinner and scientific program at the Worcester City Hospital, Worcester, Mass.

February 14—Dinner and scientific program at the Worcester State Hospital, Worcester, Mass.

March 14—Dinner and scientific program at the Memorial Hospital, Worcester, Mass.

April 11—Open date.

May 9—Annual Meeting. Time and place to be announced later.

ERWIN C. MILLER, M.D., Secretary.

27 Elm Street, Worcester, Mass.

BOOK REVIEW

Holt's Diseases of Infancy and Childhood. By the late L. EMMETT HOLT, M.D., and JOHN HOWLAND, M.D. Revised by L. Emmett Holt, Jr., M.D., Associate Professor of Pediatrics, Johns Hopkins University, and Rustin McIntosh, M.D., Carpentier Professor of Diseases of Children, Columbia University. Tenth Edition. D. Appleton and Company, New York and London. Price $10.00.

In this, the tenth edition of the famous *Diseases of Infancy and Childhood*, by Holt and Howland, now revised by a son of the original author and Rustin McIntosh, his successor as Professor of Pediatrics at Columbia University, the work easily holds its place as the leading textbook of Pediatrics published in this country. The best of the old is retained; the worthwhile of the new is added and the style continues to be an example of forceful and lucid exposition.

Those subjects in which important advances are being made have been brought up to date; as examples the anemias and hemophilia, while no unproved doctrines are accepted. A laudable conservatism is evident as is shown by a refusal to accept the calamitous thymus vogue and a healthy skepticism concerning the value of active immunization with scarlet fever toxin. The work has been enriched by the inclusion of adequate lists of references. The new editors are to be congratulated on the care and judgment which they have exercised in preparing this worthy successor to a significant line of illustrious predecessors.